

THE DIGESTIVE TRACT IN
ROENTGENOLOGY

VOLUME ONE

The Digestive Tract in ROENTGENOLOGY

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SECOND EDITION

TWO VOLUMES

1,531 Illustrations in 897 Figures

VOLUME ONE

Introduction

The Hypopharynx and the Esophagus

The Stomach

The Duodenum



Philadelphia

London

Montreal

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Second Edition

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DEDICATED TO

MY WIFE ESTHER

*whose constant encouragement and
co operation have been of inestimable
value in the preparation of this work*

Preface to Second Edition

The appearance of this new edition of the book marks the completion of almost thirty two years of affiliation with the Gastro intestinal Roentgen Division of Bellevue Hospital. The enormous amount of material in this institution, as well as that obtained through my other affiliations and in my private practice, made possible the writing of this book.

As in the past, I have kept the following criteria in mind in the choice of the material to be included in this work:

- 1 Complete documentation of the validity of the roentgenographic diagnosis as determined by operation or autopsy with histologic confirmation

- 2 The clarity of the roentgenographic deformity as noted in the illustrations

- 3 The usefulness of the material from both the clinical and the roentgenographic points of view

I have also attempted through brief case histories to intertwine the essential clinical

features with roentgenographic and pathologic findings. The technical procedures essential for the clear demonstration of the roentgenographic deformities of the particular segment involved have been stressed.

Although much rare material has been included in this book, an attempt has been made to keep the subject properly balanced, with major emphasis upon those lesions which the internist, the gastroenterologist and the radiologist are more apt to meet in their practice.

The writing of the new edition has enabled me to include a large amount of additional material in order to enhance its usefulness and also has given me the opportunity to bring the rapidly expanding literature on the subject up to date.

Again I wish to express my deep appreciation of the courtesy and the co-operation of the J. B. Lippincott Company.

JACOB BUCKSTEIN

Preface to First Edition

The diagnostic value of the roentgenographic method in the study of the digestive tract is fully recognized by internist and surgeon alike. A knowledge of its potentialities should be of great value to the general practitioner.

For more than a quarter of a century it has been my privilege to be affiliated with Bellevue Hospital. During this period I have had the opportunity of correlating the roentgenographic findings in disorders of the digestive tract with those obtained at operation or autopsy. This book is an outgrowth of my experience with the enormous amount of material at the hospital and of my own private practice.

In the pages of this book the reader will find a presentation of those technical procedures which I have found most helpful and of the abnormalities of the digestive tract, both functional and organic, in the study of which this diagnostic aid is of inestimable value.

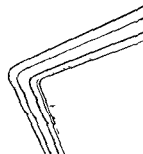
Some of the illustrations in this volume appeared in my earlier volume, *Clinical*

Roentgenology of the Alimentary Tract, published by W. B. Saunders Company, and I give them my thanks for permission to reproduce these illustrations.

I wish to express my gratitude for the excellent co-operation that I have received from the various members of the hospital staff and to the publishers, J. B. Lippincott Company, for the many courtesies extended to me.

I wish in particular to record my debt to Dr. Lewis Gregory Cole, whose inspiring lectures were my first introduction to the field of roentgenology of the digestive tract when I was a medical student at Cornell; to Dr. Russell D. Carman, whose book I read and reread while a medical officer during World War I; to Dr. I. Seth Hirsch, formerly Director of the Roentgen Department of Bellevue Hospital; to Dr. Lewis J. Friedman, the present director of the department; to Dr. Eugene F. DuBois and Dr. David P. Barr of the Cornell Medical College; and to Dr. Louis Hauswirth, Director of Medicine, Beth David Hospital.

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JACOB BUCKSTEIN

Contents

VOLUME ONE

INTRODUCTION

1 HISTORY OF ROENTGNOLOGY OF THE ALIMENTARY TRACT	3
---	---

THE HYPOPHARYNX AND THE ESOPHAGUS

2 THE HYPOPHARYNX	11
Roentgen Examination of the Hypopharynx	12
Lateral Pharyngeal Pouches	22
Myasthenia Gravis	24
Bulbar Palsy and Dysphagia	28
3 THE NORMAL ESOPHAGUS	30
Anatomic Considerations	30
Technic of Roentgen Examination	31
The Esophagus in Infancy	37
4 CONGENITAL ANOMALIES OF THE ESOPHAGUS	39
Anomalies of Position	39
Anomalies of the Aortic Arch	42
Double Aortic Arch	44
Roentgen Diagnosis of Vascular Ring Formations	46
Anomalies of Formation	49
Congenital Narrowing of Esophagus	61
5 DISPLACEMENT AND COMPRESSION OF THE ESOPHAGUS BY EXTRA ESOPHAGEAL LESIONS	67
Cardiac Disease	67
Aortic Aneurysm	68
Extra esophageal Tumor	70
6 DIVERTICULA OF THE ESOPHAGUS	76
Types of Diverticula	76
Pharyngo esophageal Diverticula	76
Traction Diverticula	81
Traction Pulsion Diverticula	83
Transient Diverticula	85
Congenital Diverticula	85
Epiphrenic Diverticula	86

7	CARDIOSPASM	95
	Roentgen Characteristics	96
8	BENIGN TUMORS OF THE ESOPHAGUS	101
	Roentgen Diagnosis	102
9	MALIGNANT LESIONS OF THE ESOPHAGUS	108
	Roentgen Diagnosis	108
10	FOREIGN BODIES IN THE ESOPHAGUS	119
	Roentgen Diagnosis	119
11	ESOPHAGEAL VARICES	122
	Roentgen Diagnosis	123
	Esophageal Varices in Children	130
12	PEPTIC ULCER OF THE ESOPHAGUS	132
	Roentgen Diagnosis	134
13	ESOPHAGITIS	139
	Roentgen Diagnosis	141
14	THE ESOPHAGUS IN SCLERODERMA AND THE COLLAGEN DISEASES	153
15	SYPHILIS AND TUBERCULOSIS OF THE ESOPHAGUS	160
	Syphilis	160
	Tuberculosis of the Esophagus	160
16	THORACIC STOMACH ASSOCIATED WITH A SHORT ESOPHAGUS	163
	Roentgen Diagnosis	165

THE STOMACH

17	THE NORMAL STOMACH	173
	Technic of Roentgen Examination	173
	Roentgen Characteristics of the Normal Stomach	174
	The Stomach in Infancy	176
	The Tonus of the Stomach	176
	Duplication of the Stomach	182
	Displacement of the Normal Stomach by Extragastric Tumors	183
	The Gastric Air Bubble	196
	Gastric Motor Function	197
	Gastrospasm	197
	The Gastric Mucosa	199
18	CHRONIC GASTRITIS	207
	Etiology and Pathology	207
	Roentgen Diagnosis	207
	Granuloma of the Stomach with Eosinophilic Infiltration	215
19	HYPERTROPHY OF THE PYLORUS	219
	Etiology	219
	Roentgen Diagnosis	220

20	GASTRIC ULCER	233
	Localization and Frequency	235
	Roentgen Diagnosis	235
	The Size of the Ulcer and Its Histopathology	267
	The Healing of Gastric Ulcer	271
	Relation of Benign to Malignant Gastric Ulcer	280
	Roentgen Diagnosis of Malignant Gastric Ulcer	280
21	DIVERTICULA OF THE STOMACH	297
	Roentgen Diagnosis	299
22	BENIGN TUMORS OF THE STOMACH	303
	Incidence and Types	303
	Roentgen Diagnosis	305
23	BEZOARS AND OTHER FOREIGN BODIES OF THE STOMACH	330
24	MALIGNANT TUMORS OF THE STOMACH	335
	Incidence and Location	335
	Roentgen Diagnosis	335
	Amyloidosis of the Stomach	341
	Neurofibroma of the Stomach	342
	Mass Roentgenography	343
25	SYPHILIS AND TUBERCULOSIS OF THE STOMACH	377
	Syphilis of the Stomach	377
	Tuberculosis of the Stomach	381
26	THE POSTOPERATIVE STOMACH	394
	Roentgen Studies Following Gastro enterostomy	394
	Gastrojejunal and Jejunal Ulcer	399
	Other Complications Following Gastro enterostomy	406
	Jejunogastric Intussusception	407
	Perforation and Fistulae	408
	Healing of Jejunal Ulcer	412
	Roentgen Findings after Other Operative Procedures	414

THE DUODENUM

27	THE NORMAL DUODENAL BULB, INCLUDING DUODENITIS	425
	Anatomy of the Duodenum	425
	Roentgen Demonstration of the Duodenum	425
	Duodenitis	426
28	DUODENAL ULCER	451
	Early Descriptions	451
	Incidence and Location of Duodenal Ulcer	451
	Roentgen Diagnosis of Duodenal Ulcer	452
	Duodenal Ulcer Beyond the Bulb	455
	Combined Gastric and Duodenal Ulcer	456
	Peptic Ulcer in Infancy and Childhood	458

mann's "mass," which consisted primarily of chalk and therefore should be as opaque to the roentgen rays as the bones. After an exposure of 57 minutes, they were able to show the blood vessels in the roentgenogram even more distinctly than the bones themselves. In a second case, they roentgenographed the little finger of the left hand of a colleague, this had been injured by a bullet and showed the presence of callus formation. They recognized in this finding a new method for the demonstration of bone pathology. The actual roentgenograms accompany the article. They wrote, "Should it be possible through technical improvement to radiograph other portions of the body, this would open up a new field in diagnosis for the healing of the sick."

An important step forward occurred with the publication of the highly interesting paper of Sehrwald.² The importance of his contribution rested on the fact that he showed that chlorine, bromine and iodine absorbed the roentgen rays and that this property depended upon the atomic weight of the elements. He predicted that internal organs filled with these halogens would become visible to the roentgen rays.

The next step in the development of the potentialities of the roentgen method was in the visualization of those internal structures of the human being, such as the esophagus, stomach and the rest of the digestive canal. This occurred with the publication of the work of Becher.³ He recorded his observations on the outline of the stomach and intestines of a guinea pig after first filling them with lead acetate. He hit upon the two great essentials on which further progress depended, namely "a solution must be employed which can be introduced into the human stomach without causing damage and which is non-transparent for roentgen rays."

Within a short time Wegele⁴ had made a suggestion which was later carried out by Lindemann,⁵ who first placed a copper wire to a rubber tube, which he then intro-

duced into the stomach of a human being, he succeeded in roentgenographing this, outlining the curvature of the stomach. He also succeeded in doing this in the case of a patient with gastric dilatation. Actual roentgenograms which required a 16-minute exposure accompany his interesting account.

Less than a month after the original contribution of Becher, Hemmeter⁶ published his paper on the roentgen visualization of the human stomach. In his original article Hemmeter, after describing the attempt of Becher to roentgenograph the stomach and a loop of intestine of a guinea pig, said "To obtain a photograph of the human stomach a solution is necessary having two properties (1) it must not injure the stomach of the subject to be photographed, (2) it must be impenetrable to the Rontgen rays." He did say, however, "I do not consider the use of the Rontgen method, on account of its complexity and long duration of exposure, as practical for determining the size and location of the stomach." While suggesting that lead acetate might be put into an elastic bag introduced into the stomach and then roentgenographed, he personally did not put the procedure to practical effect.

Rumpel⁷ then outlined a dilated esophagus by filling it with a suspension of bismuth subnitrate.

The pioneer work of Cannon in the early development of the roentgenology of the alimentary tract will forever remain a credit to American science. Because of its historic significance I quote at length from his paper, published in 1914, on the "Early Use of the Roentgen Ray in the Study of the Alimentary Canal."

In the fall of 1896, when I was a first-year student in the Harvard Medical School and Mr. Albert Moser, a Cambridge acquaintance, was a second-year student, we went to Professor H. P. Bowditch and asked for an opportunity to undertake a physiologic investigation. He suggested that we test by means of the newly discovered Roentgen rays the Kronecker-Meltzer view of deglutition, namely, that substances when swallowed are

shot down the oesophagus by pressure developed in the mouth, and are not pushed down by a peristaltic wave. A small static machine and some simple tubes were secured, and we set to work. Our first observation was made on Dec 9, 1896, when we watched globular pearl buttons pass down the oesophagus of a dog. On Dec 14, we repeated the observations on a rooster, whose neck was kept straight by fastening the head and body in fixed positions.

That substances vary in their opacity to the Roentgen ray in accordance with their molecular weight was then common knowledge. Barium and bismuth were selected from among the heavy metals, and barium sulphate and bismuth subnitrate were chosen as salts which, because practically insoluble, would serve our purposes. Since bismuth subnitrate was a pharmacopoeial preparation we determined first to use that.⁸

On Dec 16, 1896, we gave a frog a gelatin capsule filled with this bismuth salt, and after seeing clearly the shadow of the substance in the frog's stomach, we repeated, the next day, Dec 17, the use of the salt in a capsule to show deglutition in the dog. When the swallowed capsules were dissolved the bismuth subnitrate was liberated and in my notes I find the remark that thus a dark roundish area was produced as a stomach shadow. Then we procured a goose and made for it a box so arranged that the long neck reached up through the cover. A high cardboard collar was then attached to the top of the box in such a way that it could be closed in front when surrounding the goose's neck. Thus the goose with the appearance of using the most stylish neckwear, presented to the fluorescent screen a very satisfactory extent of oesophagus. At a meeting of the American Physiological Society in Boston Dec 29, 1896, the phenomenon of deglutition as exhibited by the goose when swallowing capsules containing bismuth subnitrate was informally demonstrated to the members by means of the Roentgen rays. This was I think, the first public demonstration of movements of the alimentary tract by use of the new method. Later I showed that barium sulphate mixed with the food was quite as satisfactory as bismuth subnitrate.⁸

It was not until Jan 9 1897 that we employed bismuth subnitrate mixed with food (in this case a bread mush) to render the swallowed mass visible. And in order to test the rate at which substances with different consistencies move along the oesophagus we added more or less water to the mush thus

varying its physical state from a rather stiff mass to a very soft nearly fluid mixture. Various observations of this nature were made on the goose in January, February and March 1897. On April 3 1897, we began using the same mush bismuth bolus and also meat with bismuth subnitrate adherent, in a study of swallowing in the cat, and as the mixture of bismuth subnitrate and food accumulated in the stomach that organ was outlined by a dark shadow. On April 3 and in observations on several days thereafter, the gastric contents became visible as the cats swallowed more and more of the mixture, but no movements of the wall were seen. Finally on April 23, 1897, a cat was fed bread soaked in warm water, and mixed with bismuth subnitrate about an hour and a half later peristaltic waves were clearly seen passing over the organ.

At a meeting of the American Physiological Society in Washington, May 4, 5 and 6, 1897, a preliminary report of these observations was presented by Professor Bowditch, and the summary of the report was published a month later (*Science*, June 11, 1898, p 901). A part of the summary was as follows:

For this purpose (studying the oesophageal and gastric movements by means of Roentgen ray shadows) moist bread meat, mush or viscid fluids were mixed with bismuth subnitrate. Food thus prepared is visible during the process of deglutition, and if given in sufficient quantities serves to outline the stomach and to render its peristaltic movements visible. Then followed an account of the results which had been obtained in observing deglutition in different animals and also gastric peristalsis.

Roux and Balthazard⁹ in June 1897, described a series of experiments on the motor function of the stomach of various animals by means of the Roentgen ray by employing a mixture containing bismuth subnitrate. In July of that year they gave a brief report of their experiences with the method in studying the contractions of the stomach of man. In a later publication they¹⁰ continued their studies on the movements of the stomach by the Roentgen method with the administration of bismuth subnitrate and presented actual radiographs. They concluded their study by saying:

The stomach is divided into two distinct regions: the largest part of the stomach serves

as a reservoir for food, the prepyloric portion is the motor region of the stomach, and by its vigorous peristaltic movements, it forces into the duodenum, little by little, the material accumulated in the stomach

In 1898, Cannon¹¹ published the results of his research on the behavior of the esophagus, during which experiments he recorded his findings in the case of a girl of 7 years to whom he administered a suspension of bismuth in water. That same year and in the following year, 1899, Cannon worked with Dr. Francis H. Williams in the roentgen visualization of the stomach of a boy aged 10 and that of his 7-year-old sister. They were given a meal of a pint of milk and bread to which was added 30 Gm of bismuth subnitrate. After the outline of the stomach was visualized on the fluorescent screen, drawings were made on transparent paper placed over the gastric image. These results were published in 1903.¹²

Rieder¹³ in 1905 recommended a mixture of flour or potato with bismuth subnitrate for the visualization of the alimentary tract. He recommended the use of 30 Gm of bismuth by mouth.

Of historical interest is the original contribution of Holzknecht and Brauner.¹⁴ They presented a detailed description of the technic of fluoroscopic study of the bismuth-filled stomach. In some cases they not only administered a suspension of bismuth but introduced in addition an effervescent mixture to produce gaseous distention of the stomach. Included in this contribution are drawings of the appearance of the stomach under different conditions. Of interest also is a photographic reproduction of the laboratory, showing a picture of Holzknecht and a number of other doctors in front of the fluoroscopic screen observing a patient who is being examined.

Holzknecht and Jonas carried roentgen exploration still further in their contribution of 1909.¹⁵ They described their combined fluoroscopic and roentgen procedure in the examination of the stomach and

duodenum and stressed the importance of palpatory manipulation. The patient, placed behind the screen with his stomach empty, was examined following the administration of 10 to 15 Gm of bismuth carbonate in 100 cc of water. The passage of the opaque suspension was followed through the esophagus into the stomach and was then forced manually into the pylorus and duodenum. They studied the form, position and peristalsis of the stomach and emphasized the normal nature of the air in the stomach, the presence of which in the pre-roentgen era had been attributed to nervous phenomena. They emphasized the significance of fluoroscopy in the study of the anatomy and physiology of the normal stomach and stressed its diagnostic value in the demonstration of nonpalpable tumors, pyloric stenosis, carcinoma, adhesions and malfunction of a gastroenteric stoma.

So remarkable were the potentialities of this new method of diagnosis that H. Bécélère,¹⁶ envisioning the possibilities of the future, said

The introduction into the stomach of a very large quantity of bismuth in a mixture of bread and milk has enabled Roux and Balthazard in France and Williams and Cannon in America to make interesting researches in animals, children and adults on the muscular function of the digestive canal. It is to be hoped that clinical medicine will take part in these researches and that in this manner visceral radiology will some day be enriched by a new chapter.

That the hope of Bécélère has met with rich fruition is demonstrated by the accuracy which the roentgen method has achieved and the reliance placed by the clinician and the surgeon upon the objective significance of the information which is obtained.

In the following pages of this book will be unfolded the story of the achievements in diagnostic roentgenology of the alimentary tract, which have fully justified the prophecy of Bécélère. Much of this has depended on the elaboration of technical aids

in the construction of apparatus, and an incalculable debt is due the physicists and the engineers who have contributed to this progress. No attempt will be made in this work, however, to discuss details regarding the structure of roentgen apparatus, tubes, cassettes, superspeed screens or any of those other features which can best be learned from actual contact with such apparatus or from books specifically devoted to the subject of technic or in a course in the physics of roentgenology—best learned in conjunction with the subject of general roentgenology itself.

Such details as concern the preparation of the patient, the nature of the mixture recommended for administration to the patient, the position of the patient, the time interval between exposures and similar points of a clinical character, however, will all be discussed in relation to the specific phase of the alimentary tract under investigation—each in its appropriate chapter.

One feature of a technical character which I do wish to emphasize is the use of a Bucky diaphragm attached to the fluoroscopic screen. I consider this added feature of inestimable value in affording a clearer and more detailed visualization of the image, and I strongly recommend that it be made a permanent addition to the fluoroscopic screen in the study of the alimentary canal.

The modern fluoroscope is constructed in such a manner as to give a maximum degree of safety in diagnostic exploration. Various spot film devices are now obtainable for graded compression of an isolated area of the alimentary tract, with a switch over for prompt radiographic exposure of the region under special observation. If in addition to the safety devices constructed within the fluoroscope itself for the protection of the operator a chair is employed with a lead protective apron in front of soft lead lined gloves are used and if intermittent exposure is made of as small an image as is consistent with proper visualization of the various portions of the visc-

being studied, the procedure may be considered essentially safe.

At present, the question as to whether one should depend on fluoroscopy or upon roentgenography in the examination may be considered obsolete. The combination of both procedures, fluoroscopy and roentgenography, is essential in order to obtain the maximum of detail of a diagnostic nature in the roentgen examination of the alimentary tract. Both procedures complement each other and there is peril in the entire elimination of one or the other. Certainly, fluoroscopy will give extremely valuable information regarding the grosser details of structure and the kaleidoscopic changes of physiologic behavior. Many lesions, even though small, will be picked up by the expert on fluoroscopic examination alone if the patient is thoroughly examined through every angle and if manual compression is employed to bring out the mucosal relief. For the finer changes of structural detail the radiograph has its advantages. Maximum safety in diagnosis consists, therefore, in the employment of both procedures in every case.

Moreover, the roentgenogram serves as a permanent record of the findings and is free of the personal equation involved in a drawing of an evanescent fluoroscopic image. Such an objective record as the roentgenogram can be studied at great length, in leisurely fashion, at any time, and long after the fluoroscopic observation has been completed. It may serve for teaching purposes and may be placed in a viewing box in the operating theater as a guide in cases of surgical intervention. A record of this kind can also be used for purposes of comparative study in the determination of the effectiveness of medical management of any particular disorder and, as in the pages of this book, such roentgenograms can be used for purposes of publication. No matter how skillfully trained the gastro-enterologist or radiologist may be, there are obvious advantages in the correlation of both fluoroscopy and roentgenography in the examina-

tion of any patient in whom a lesion of the gastro-intestinal tract is suspected

Finally, I wish to stress the importance of the clinical background of every such case that is studied radiographically. While the objective roentgen findings in many cases are of such a clear-cut character that their diagnostic significance may be accepted unequivocally, there are not infrequently borderline cases in which only the interplay of all the factors that can be brought to bear, whether clinical, pathologic or radiologic, will enable us to render maximum diagnostic service to our patients.

REFERENCES

1

Haschek, E, and Lindenthal, O Ein Beitrag zur praktischen Verwerthung der Photographie nach Rontgen, Wien klin Wchnschr 9 63, 1896

2

Sehrwald, E: Das Verhalten der Halogene gegen Rontgenstrahlen, Deutsche med. Wchnschr 24 477, 1896

3

Becher, W · Zur Anwendung des Rontgenschen Verfahrens in der Medizin, Deutsche med Wchnschr. 22 202, 432, 1896

4

Wegele, C Ein Vorschlag, zur Anwendung des Rontgenschen Verfahrens in der Medizin, Deutsche med Wchnschr 22 287, 1896

5

Lindemann, E Demonstration von Rontgenbildern des normalen und erweiterten Magens, Deutsche med Wchnschr 23 266, 1897

6

Hemmeter, J C Photography of the human stomach by the Rontgen method a suggestion, Boston M & S J 134 609, 1896

7

Rumpel, T Die klinische Diagnose des spindelformigen Speiserohrerweiterung, Munchen med Wchnschr 44 383, 420, 1897

8

Cannon, W B · The passage of different foodstuffs from the stomach and through the small intestine, Am J Physiol 12 387, 1904

9

Roux, J, and Balthazard, V Sur l'emploi des rayons de Rontgen pour l'étude de la motricité stomacale, Compt rend Soc biol 4 567, 1897

10

———, and ———. Étude du fonctionnement moteur de l'estomac, Arch de physiol norm et path (ser 5) 10 85, 1898

11

Cannon, W B, and Moser, A The movements of the food in the esophagus, Am J Physiol 1 435, 1898

12

Williams, F. H. The Roentgen Rays in Medicine and Surgery, ed 3, New York, Macmillan, 1903.

13

Rieder, H Beiträge zur Topographie des Magen—Darmkanales beim lebenden Menschen nebst Untersuchungen über den zeitlichen Ablauf der Verdauung, Fortschr Geb Rontgenstrahlen 8 141, 1905

14

Holzknecht, G, and Brauner, L Die radiologische Untersuchung des Magens, Wien klin Rundschau 19 273, 311, 332, 364, 399, 1905

15

Holzknecht, G, and Jonas, S Die Rontgenuntersuchung des Magens und ihre diagnostischen Ergebnisse, Ergebn inn Med u Kinderh 4 455, 1909

16

Béclère, H Exploration radiologique de l'abdomen, Arch d'électr méd 10 651, 1902

THE HYPOPHARYNX
AND
THE ESOPHAGUS

2

The Hypopharynx

The act of swallowing is a complicated reflex initiated by a chain of physiologic factors in the pharyngeal region. The pharynx is a musculomembranous organ about 5 inches in length, which extends from the base of the skull to about the level of the sixth cervical vertebra, where it becomes continuous with the esophagus. By virtue of its communications anteriorly it is divided into a nasal pharynx, an oral pharynx and a laryngeal pharynx. The laryngopharynx extends from the laryngeal opening to the cricopharyngeal constrictor. It is thus bounded above by the epiglottis below which is the superior aperture of the larynx. Laterally are the pyriform sinuses. Medial to the sinuses are the aryepiglottic folds, while laterally are the thyroid cartilage and the hyothyroid ligament. Above the epiglottis are two other recesses, the valleculae. The pharyngoepiglottic folds are at the lateral margins of the valleculae and extend from the epiglottis to the pharynx.

The bolus of masticated food is passed through the isthmus of the fauces by the action of the anterior portion of the tongue against the palate. This is also aided by the posterior portion of the tongue forcing the bolus through the pharynx into the esophagus.¹ The bolus is passed through the pharynx into the esophagus in about one fifth of a second from the time of contraction of the mylohyoid muscles. This complicated act is controlled by a center in the medulla, with the effectors present in the muscles of the larynx and the pharynx. During this process the oral and the nasal cavities are closed off and the larynx is occluded by the adduction of the vocal

cords and the elevation of the larynx. When because of any disorder there is interference with the elevation of the larynx, swallowing is impossible.

The junction of the hypopharynx and the esophagus was described by Killian² in 1908 on the basis of endoscopic studies. He described a transverse fold on the posterior wall of the esophagus at its junction with the hypopharynx. Ordinarily this fold occluded the opening of the esophagus but this region relaxed on swallowing and when a vomiting reflex was produced. He called this area the 'mouth' of the esophagus and called the bulge on the posterior wall the esophageal 'lip'. He correlated the anatomic localization of the lip with the transverse portion of the cricopharyngeus muscle.

That the indrawing of the posterior wall of the pharyngo esophageal junction is due to the activity of the transverse portion of the cricopharyngeus muscle is supported by the following observations:

- 1 In favor of the assumption that this smooth indentation is due to the functional behavior of the cricopharyngeus muscle is the fact that pulsion diverticula originate in this region. It is a generally recognized anatomic fact that these diverticula originate on the posterior wall of the pharyngo esophageal junction in the weak area between the oblique and the transverse fibers of the cricopharyngeus muscle. Since the smooth indentation on the posterior wall corresponds to the area just below the origin of these diverticula, there is added confirmation that the concavity is produced by the action of the transverse portion of this muscle.

2 In studying the roentgen appearance of this concave indentation one may note that there is considerable variability in the degree of constriction in this region and in the length of the segment involved. At times, this area may be completely shut off, as in marked dilatation of the esophagus in cardiospasm or even in carcinoma, thereby preventing regurgitation into the pharynx and ultimately into the tracheobronchial tree. One may best explain such a phenomenon as being due to constriction produced by a muscular structure capable of alteration in its physiologic behavior, now yielding and now causing occlusion to a variable degree. The anatomic localization of this indentation, as well as its behavior and its association with the origin of the classical pulsion diverticulum in this region, all point strongly to the transverse portion of the cricopharyngeus muscle as the cause.

ROENTGEN EXAMINATION OF THE HYPOPHARYNX

While the pharynx and the pharyngo-esophageal junction may be noted sometimes after a simple swallow of barium, the passage of the opaque fluid is often so rapid as to make careful visualization of this region difficult or impossible. One may caution the patient not to repeat the swallowing act after the first mouthful of barium has filled out the pharynx, thus permitting some of the barium to outline the contour of the pharynx as well as the valleculae and the pyriform sinuses. An aid in arresting the barium for a more protracted period of observation is the Valsalva test. This may be carried out as follows. The patient is told to take a deep breath. Before expiration he keeps his mouth closed tight and his nose is pinched. He is then instructed to exhale. As a result, the increased intrathoracic pressure transmitted by way of the trachea distends the pharynx with air. In this manner, the barium within the recesses of the pharynx may not only be retained but will stand out in sharp contrast. In

many cases, however, the Valsalva method is ineffectual in delaying the rapid emptying of the pharynx.

Retention of barium in the pharynx and in the pharyngeal recesses will in part depend on the nature of the mixture, since obviously a thick paste will cling to these regions more readily than a simple barium-water suspension. The method of preparing the thick mixture will be described in connection with the roentgen examination of the esophagus. The visualization of the pharyngeal structures will also depend on the prompt fixation in the roentgenogram at the optimum moment of exposure, usually after the first swallow of a small amount of the thick mixture and before subsequent swallowing has dislodged the barium. Spot-film technic is an invaluable aid. Examination in the supine position may also be helpful in retarding the flow of barium. In some cases, there may be retention of barium in the recesses without either subjective complaints or evidence of organic derangement as shown by esophagoscopy examination. In other cases, however, with such retention, particularly when the quantity is marked and the period prolonged, actual organic changes may be noted, some of which are recognizable on roentgen examination.

As viewed laterally the wall of the hypopharynx when distended with barium may show a slight superficial irregularity of its anterior surface. The posterior wall is smooth and often exhibits wavelike ripples apparently due to peristaltic activity. The more generally recognized alteration is on the posterior wall at its junction with the esophagus and appears as a smooth, rounded indentation, the anatomic basis for which has already been described. The narrowing at the junction of the pharynx and the esophagus may at times produce practically complete occlusion because of the functional behavior of the cricopharyngeus muscle. This may act as a protective mechanism by preventing regurgitation into the pharynx and the trachea in the presence

of an obstructive lesion of the esophagus of either organic origin or the result of derangements of function

Viewed anteriorly, the pharynx when distended with barium, is of smooth contour, usually tapering gradually at its junction with the esophagus. After partial emptying of the pharynx, rests of barium may be noted, outlining the valliculae which appear as two small, cup shaped areas to either side at about the level of the thyroid cartilage. Below are the bilaterally placed, triangular shaped pyriform sinuses. In the lateral view one or another of these structures may be seen but often only with difficulty or not at all, because they may overlap each other.

Abnormalities of the hypopharynx may result from a number of causes, both functional and organic. Faulty coordination of the swallowing mechanism, with failure of relaxation of the cricopharyngeus muscle at the proper moment, may well be a cause of transient dysphagia of functional origin. The only roentgen evidence of such lack of coordination may be abnormal delay in the emptying of the recesses of the hypopharynx, as well as unusual prominence of the concavity produced by the pressure of the transverse portion of the cricopharyngeus muscle with occlusion at the pharyngo-esophageal junction. The more carefully this region is studied, however the fewer are the cases of dysphagia of a purely functional nature. The most serious cause of intrinsic origin is carcinoma of the hypopharynx. Similarly foreign bodies caught at the mouth of the esophagus may cause obstruction. A pulsion diverticulum when large, may compress the pharyngo-esophageal junction. This will be discussed more fully in the chapter on diverticula of the esophagus.

A comparatively rare but clinically important disorder is the dysphagia occurring mainly in middle aged women and associated with secondary anemia, a condition which originally had been considered hysterical and still is referred to occasionally

as "globus hystericus." The association of anemia with this condition was emphasized by Blankenstein³ in 1893 in whose dissertation will be found references to the older literature on the subject. Plummer in 1914 and Vinson in 1922 thought that the dysphagia was purely functional, although Pater-son⁴ in 1906 had described atrophic changes in the pharynx as a concomitant finding in this syndrome.

That organic changes of the pharyngo-esophageal region are present in this disorder was shown also by Mosher who described the presence of webs at the entrance to the esophagus behind the cricoid cartilage. They appear as thin folds of mucous membrane and scar tissue, roughly crescentic. While the small web may be present without symptoms unless food is eaten unusually fast, the large webs may be a cause of definite obstruction and may be the underlying organic process in individuals stamped with the diagnosis of globus hystericus.

The presence of organic changes in the hypopharynx and the upper esophagus in association with the syndrome of dysphagia and anemia has been corroborated by a number of observers. Kelly,⁶ Paterson⁷ in his later article, Suzman⁸ and Hoover⁹. One of the cases of dysphagia and anemia reported by McGee and Goodwin¹⁰ was studied at autopsy. The upper portion of the esophagus was hyperemic and showed irregular areas of denudation with islands of lymphocytes and scattered plasma cells. The muscular wall was thickened evidence of a chronic esophagitis.

There is therefore ample evidence of an underlying organic basis for this type of dysphagia associated with anemia.

Of great significance is the apparent predisposition of these organic alterations to the ultimate development of carcinoma of the hypopharynx (Pater-son, Ahlbom¹¹ and Simpson¹²).

Many of the changes may be recognized on radiologic examination of the hypopharynx (Mosher and Waldstrom and



FIG. 4 The constriction produced by the transverse portion of the cricopharyngeus muscle is particularly well shown in the above illustration

constriction at the junction of the hypopharynx and the esophagus may give rise to dysphagia

Illustrative Case. I G, male, aged 62 The patient, a known cardiac, was admitted to the hospital complaining of inability to swallow even fluids and of vomiting of 3 days' duration

Three years previously he had had a similar episode of dysphagia He had lost 45 pounds in the preceding 4 years Physical examination revealed evidence of auricular fibrillation His hemoglobin was 90, R B C 5 200,000

Roentgen examination at this time (Fig 5) revealed obstruction in the pharyngo-esophageal region with reflux of the barium by way of the pharynx into the trachea. The evidence was of an obstructive lesion in that region

On esophagoscopy the instrument was passed with ease into the stomach No obstruction or foreign body was seen nor was any other explanation of the symptoms found Neurologic examination was negative.

After 4 days, the patient's dysphagia suddenly disappeared and he was able to drink



FIG 5 Functional obstruction at the pharyngo-esophageal junction



FIG 6 Obstruction of the cricopharyngeal region by a prune pit



FIG 7 Note the linear opacity at about the level of the sixth cervical vertebra produced by the chicken bone



FIG 8 Carcinoma of the hypopharynx. Note the narrowing and distortion of the hypopharynx and the superficial irregularity of the anterior wall

liquids and even eat solid foods. Nine months later, the patient returned because of dimness of vision and colored scotomata during the preceding 3 to 4 days. His cardiac symptoms were well controlled by digitalis. He had no dysphagia at this time.

The fact that he had had two episodes of dysphagia 3 years apart, the negative findings on esophagoscopy, the short duration and abrupt termination of the second attack with complete relief of the dysphagia thereafter, all point strongly to a functional derangement as the cause, probably in the nature of an extreme degree of cricopharyngeal spasm with total constriction of the lumen in this region.

Obstruction of the cricopharyngeal region by a prune pit is shown in Figure 6. The narrowing of this area by the action of the transverse portion of the cricopharyngeus muscle probably contributed to the arrest of the prune pit in this region.

The roentgenographic appearance of a chicken bone stuck in the distal portion of the hypopharynx may be noted in Figure 7. The patient, J. W., male, aged 49, gave a history of swallowing a chicken bone which became lodged in his throat, causing dysphagia. On esophagoscopy examination the chicken bone was found lying anteriorly across the lumen of the esophagus and embedded medially and laterally. It was removed by grasping forceps.

Malignant infiltration of the hypopharynx is illustrated by the next case.

A. M., female, aged 69. During the preceding 3 months the patient had been complaining of progressive dysphagia, at first with solid food only but at the time of her admission to the hospital with liquids as well. She had lost considerable weight.

Endoscopy disclosed a mass which bled easily. A biopsy was taken.

The pathologic report was squamous-cell carcinoma.

The roentgen examination (Fig. 8) showed an irregular, tubular narrowing of the hypopharynx, which was interpreted as representing an infiltrating lesion in this region. It is interesting to note that there had been no history of difficulty in swallowing prior to the onset of the brief period of the dysphagia which brought the patient to the hospital. There was no clinical evidence to indicate

that the carcinoma was etiologically related to any preceding underlying pathology of the hypopharynx.

The roentgen findings in tumor of the hypopharyngeal region may be noted in the following case.

G. H., male, aged 63. During the preceding 2 months the patient stated that he had had difficulty in swallowing and had had a chronic cough. When he attempted to swallow food, some of it regurgitated back through his nose. He was very hoarse. Physical examination disclosed a firm fixed mass at the lateral end of the hyoid bone on the left side, anterior to the sternocleidomastoid muscle and attached to the deeper structures and not to the skin. The mass measured 4 x 3 cm, was not tender or cystic. There was a smaller mass above this, which was also fixed but more movable than the larger mass and seemed to be a node. There was a firm nodular mass attached to the left palatopharyngeal fold, which appeared to extend posteriorly as well as anteriorly and to push the fold forward.

Laryngoscopic examination disclosed a large ulcerating mass in the left postpharyngeal wall, measuring about 3 x 2 cm, with a large crater in the center. The mass appeared to extend along the lateral pharyngeal wall to the post-tonsillar pillar and the base of the tongue. A biopsy taken from the lateral pharyngeal wall showed squamous-cell carcinoma.

Roentgen examination (Fig. 9) revealed a defect of the left lateral wall of the hypopharynx with displacement to the right and an obliteration of the left pyriform sinus.

The diagnosis was tumor of the left hypopharyngeal region.

A carcinoma of the hypopharynx may be characterized by the presence of translucent areas produced by the tumor.

C. D., female, aged 62. The patient gave a 1-month history of difficulty in swallowing and of the feeling of a lump in her throat. She was able to swallow only small amounts of liquid. At times the liquid would be forced out through her nose. On one occasion she brought up a small amount of fresh blood.

"Using a number 16 laryngoscope the pharynx was explored and almost immediately a large fungoid mass was encountered, involving part of the right and almost all of the left arytenoid. This mass extended downward and

pharynx and the entire oral hypopharynx and epipharynx. He was then irradiated with the cobalt-60. The radiotherapy was of a total dose of 4000 r.

The distance from the epipharynx to the hypopharynx was 4 cm.

Examination with the (10) x-ray tube of the epipharynx showed a normal area of the epipharynx but the rest of the hypopharynx was not visible.

After the irradiation of the epipharynx and the hypopharynx with the cobalt-60, the distance from the epipharynx to the hypopharynx was 4 cm.

A S. male, aged 40, presented with a 10 x 10 cm. area of the epipharynx and the hypopharynx. The distance from the epipharynx to the hypopharynx was 4 cm.

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Figure 1. (A) Normal hypopharynx in the left image. (B) Hypopharynx with a large tumor in the right image.

Figure 2. (A) Normal hypopharynx in the left image. (B) Hypopharynx with a large tumor in the right image.



20



FIG 11 (A, *Top, left*) Note the marked constriction with irregular narrowing produced by the carcinoma of the hypopharynx (B, *Bottom*) Malignant involvement of the hypopharynx. Note (1) the constriction of the lumen and (2) the marked forward displacement of the trachea produced by the tumor (C, *Top, right*) Carcinoma of the hypopharynx as visualized by tomography. Note the occlusion of the lumen produced by the invading tumor.

masses were palpable in the neck. A biopsy taken from the cervical mass was reported as epidermoid carcinoma. Note the deformity produced by the tumor and the occlusion of the lumen (Fig 11 C).

A benign inflammatory lesion involving the hypopharynx is illustrated by the following case:

A S, male, aged 56. The patient had been well until 9 years before hospitalization, when he developed a generalized, progressive rheumatoid arthritis involving all the joints. During the preceding 7 months he noted a progressive dysphagia so that he was finally able



FIG 11 D Chronic granulomatous inflammatory lesion of the hypopharynx. Note the smoothly outlined intraluminally placed translucent area produced by the lesion. The tracheotomy tube may be seen near the bottom of the illustration.

to swallow only liquids. He lost 35 pounds in the preceding year. Examination revealed an infiltrated lesion of the mouth, the tongue, the glottis, the larynx, and the esophagus. Repeated biopsies taken from the lesion in the larynx showed a chronic granulomatous inflammation with multinucleated giant cells. He later developed a generalized lymphadenopathy and subcutaneous nodules over the skull and the chest wall. Radiation therapy produced a decrease in the size of the axillary nodes but not of the neck nodes.

Streptomycin, nitrogen mustard, potassium iodide, penicillin, aureomycin, and sulfonamide therapy were ineffective. The chest nodules



FIG 12 Note the indentation of both the anterior and the posterior walls produced by the crescentic stricture near the hypopharyngo-esophageal junction.

broke down, and the patient developed multiple draining sinuses. Cultures were negative for acid fast bacilli and fungi. Repeated sputum examinations were negative. Biopsy of the chest nodules revealed only a granulomatous lesion. Tracheotomy was performed because of episodes of dyspnea and cyanosis. His course was downhill in spite of all the therapeutic procedures.

The final diagnosis was of a granulomatous lesion of unknown etiology.

Roentgenographic examination (Fig 11 D) was reported as follows: "Examination reveals a smoothly outlined, translucent intraluminally placed area within the confines of the hypopharyngo-esophageal junction. The evidence is that of a space-occupying lesion in the region described. The smoothness of the area of involvement is not of the type usually found in malignant infiltration."

As noted in the history, repeated biopsies taken from the hypopharyngeal region were reported as showing a chronic granulomatous inflammation. There was never any evidence of malignancy.

Evidence of the roentgen deformity produced by a crescentic stricture of the hypopharyngo-esophageal area may be noted in Figure 12.

D D, female, aged 22, gave a history of a piece of meat's being stuck in the throat on two occasions once 2 years before admission to the hospital, the second time, 3 weeks. Each time the piece of meat was removed by way of the esophagoscope. There was a history also of the patient's having swallowed lye when 5 years old.

A Robert's scope was passed down just to the cricopharynx, bringing a concentric stricture into view. Bougies were passed through this area for dilatation. The exact relationship of the swallowing of lye to the development of the concentric stricture in the hypopharyngo-esophageal region is not entirely clear, since the patient remained symptom free from the age of 5 until 14 years later, although the possibility, of course, cannot be entirely excluded.

LATERAL PHARYNGEAL POUCHES

This condition appears to be quite rare and develops apparently on a congenital basis. The explanation for their origin must be sought in the embryologic transformations which occur in the development of the branchial apparatus.

Rathke (1825) studied the branchial apparatus in the embryo of the pig. Von Baer (1827) described four branchial clefts in the human embryo. The clinical significance of the branchial clefts in relation to cysts and fistulae of the neck was described in 1832 (Ascherson). The classic work of His appeared in 1881. Working with human embryos, he made reconstructions of their development by means of wax models. His major contribution was in the description of the exact anatomic position of the branchial arches of the human embryo. The first arches meet in the midline. The lower arches show a gradual separation from one another, thereby forming a triangle which His called the "mesobranchial field."

The most thorough study of the subject of the embryology of the branchial apparatus is that of Wenglowksi.¹⁵ In the 2.6 mm embryo only the first and the second branchial arches may be noted with an indication of the first cleft. Even at this stage a sagittal section shows beginning evidence

of the branchial pouches running in the same direction as the outer branchial clefts. By the time the embryo is 6.5 mm all four arches and clefts may be noted. Wenglowksi shows a model of the embryo at this stage with the posterior portion of the head and the pharynx removed so that the anterior pharyngeal wall may be seen. Under these circumstances, one may note the four bilaterally placed pharyngeal pouches with the intervening pharyngeal arches.

By the time the embryo is 8 mm in length there are 6 arches on the left side and only 5 on the right side. From then on there is a gradual regression of these structures so that in the 11-mm embryo there are only 3 arches. The pharyngeal pouches have become shallower. In the 12-mm to 13-mm embryo only the first and the second arches are present. The pouches by this time are no more than small shallow grooves. In the normal 14-mm embryo all evidence of the branchial apparatus has disappeared. In this elaborate manner Wenglowksi traced with remarkable detail the development and the ultimate disappearance of the branchial apparatus.

The most common anomalies resulting from maldevelopment of the branchial arches and clefts are the branchiogenic cysts and fistulae of the neck. These fistulae may be of three types.¹⁶ (1) They may be complete with an external opening above the clavical and an internal opening into the pharynx near the lower portion of the tonsillar fossa. (2) The fistula may be incomplete with only an external opening and no direct opening into the pharynx. (3) The fistula may communicate with the pharynx without any external opening. Cysts may occur anywhere in the course of these fistulae. Frequently, direct injection of the fistulae with material opaque to x-rays will show the direct communication with the pharynx.

The least common type is the incomplete internal fistula with only an opening into the pharynx and none externally.¹⁷ The anatomic communication of a cyst of the

neck with the pharynx was brought out by the clinical observation of Douglas.¹⁸ He described the case of a patient 45 years old who by holding his mouth and nostrils shut and blowing hard could distend the right side of his neck with air. By pressing upon this area he was able to force the air back into the pharynx, where it escaped with a gurgling sound.

The congenital origin of cysts and fistulae of the neck has been amply corroborated clinically.^{16, 19, 4}

The lateral pharyngeal pouches of adult life may be explained on the basis of the persistence of the pharyngeal pouches which ordinarily disappear completely in the course of normal embryologic development. Certainly the anatomic predisposition for their formation is present. They may be considered as an anomaly of development of the branchial apparatus. Therefore, in addition to the cysts and the fistulae of branchiogenic origin one may include the persistent pharyngeal pouch as another example of the deviation from the normal life history of the branchial apparatus.

In the case reported by Godlee and Bucknall they described a pharyngeal pouch confirmed by operation. The story was that of a male, aged 31, who had a swelling of the left side of the neck at the level of the hyoid bone in front of the sternocleidomastoid. The size of the tumor was definitely diminished by pressure over it. On holding his breath and blowing he could inflate the tumor slightly.

At operation a pouch was removed. The narrow pedicle of the pouch passed through the thyrohyoid membrane but careful probing failed to demonstrate the actual communication with the pharynx.

The authors include an excellent bibliography of the previous literature on the subject of pharyngeal pouches and fistulae. At the time of the publication of their paper they found records of 200 such cases. They noted that the internal orifice when present was always found in the pharynx. That these lesions were of congenital origin was

strongly supported by embryologic and anatomic studies.

Raven⁶ in his discussion of pharyngeal pouches described the case of an infant 3 weeks old that was born with a swelling on the left side of the neck which caused choking attacks during feeding. He included a reproduction of the specimen obtained at autopsy which is in the Museum of Great Ormond St. Hospital for Children. This shows a large pouch on the left lateral aspect of the pharynx opening by a duct into the bottom of the left pyriform fossa. Raven considered it to be a derivative of the third endodermal pharyngeal pouch.

The persistence of these pouches may be shown occasionally by radiographic examination of the pharynx. At times these pouches may be a cause of dysphagia.²⁷ This is illustrated by the following case.

H. L., male, aged 54. For 3 years prior to examination, the patient complained of food sticking in his throat. He stated that he had to swallow "real hard to get it down." He had more difficulty in attempting to swallow solid foods but occasionally he also had difficulty with liquids. At times he might be free of dysphagia for as long as one month. He had been gaining weight. There was no vomiting. Fourteen years before, he had been operated on for a perforated peptic ulcer. Except for a mild recurrence 7 years later he remained free of gastric symptoms.

Figure 13 A shows the appearance of the hypopharynx when completely distended with barium. At this time only the pharyngeal pouch extending laterally on the right side is demonstrable.

In Figure 13 B both the right and the left lateral pharyngeal pouches may be noted. Only by means of spot film technic was it possible to demonstrate the presence of this condition. So rapidly did the entire hypopharynx empty itself of barium that it was impossible to demonstrate the structural characteristics of this region in any other manner. Of course this does not necessarily imply that food particles would escape being lodged within these diverticula and thereby cause the symptoms of which he complained.

Dysphagia associated with pharyngeal pouches is also illustrated by the next case.

H. K., male, aged 45. The patient stated

that during a period of 20 years pills "stuck in his throat." During the same length of time he frequently brought back undigested food, sometimes 3 days after eating. In addition, the patient gave a peptic ulcer history of 20 years' duration, with operation 11 years previously for a perforated duodenal ulcer. He had lost 26 pounds in the preceding 3 years.

Figure 14 A shows a large pouch originating from the right lateral border of the hypopharynx and apparently two very small ones originating from the left lateral border.

Figure 14 B With a smaller amount of barium one may note the right lateral pouch. The small ones on the left side are not demonstrable at this time. The valleculae are outlined by barium. Barium was present in the right lateral pouch for several hours. This would readily explain why pills stuck in his throat and why at times he regurgitated undigested food.

Figure 15 is an unusual example of bilateral pouches of the pharynx. The one on the right side appears to be associated with a secondary diverticulum originating from it. Although both the pouch and the secondary diverticulum may have been of congenital origin, it is also possible that the diverticulum may have been of later devel-

opment as a result of intrapharyngeal pressure transmitted to the pouch over a period of years. From that point of view it might be considered as a pulsion diverticulum originating through the wall of the congenital pharyngeal pouch.

MYASTHENIA GRAVIS

Dysphagia due to a disturbance of pharyngeal function may be a complication of myasthenia gravis. This disorder involves the muscles of the skeletal system. The muscles of the heart and of the viscera are not involved. It is characterized by weakness and easy fatigue. The muscles of the arms and the legs may be so weak as to make the lifting of the extremities difficult or impossible. In addition, the disorder may be characterized by drooping of the lids, a face devoid of expression, and in advanced cases paralysis of the respiratory muscles may lead to death. Of particular significance in so far as the digestive tract is concerned is the fact that sometimes the disorder may be characterized by involvement of the muscles of the pharynx, thereby causing

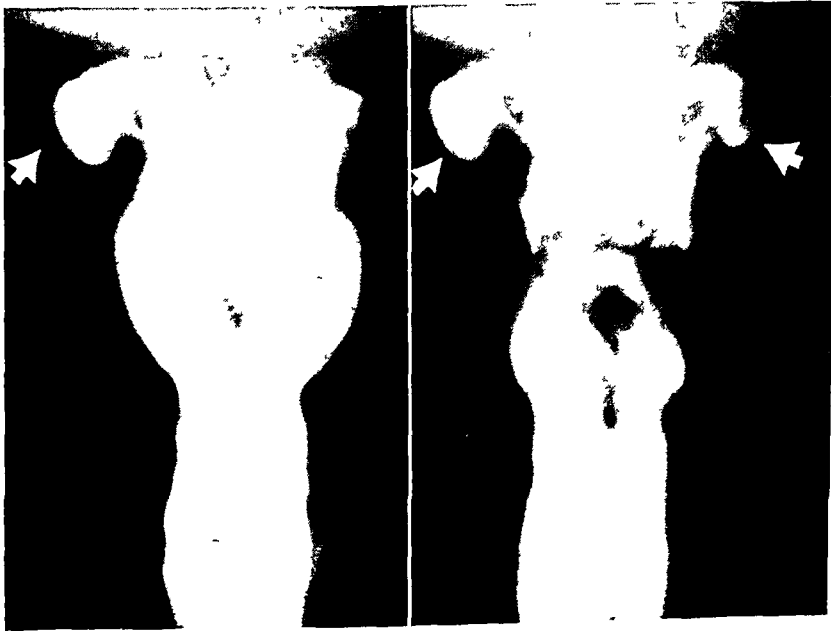


FIG 13 Bilateral pouches of the hypopharynx. (A, *Left*) Only the pouch on the right side is shown at this time. (B, *Right*) Both the right and the left lateral pouches are now demonstrable.

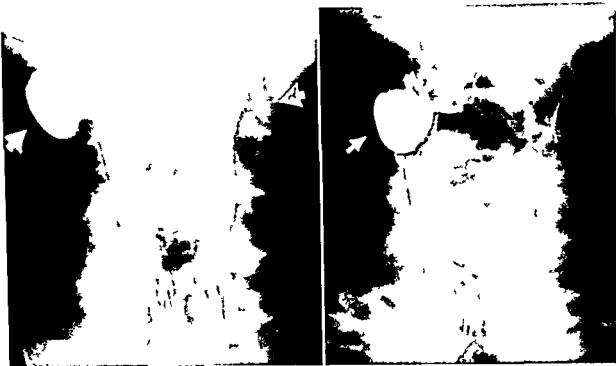


FIG 14 Lateral pharyngeal pouches (A *Left*) Note the large right lateral pouch of the hypopharynx and the two smaller pouches extending from the left lateral border. Food particles lodging in the e pouches appeared to be the cause of the dysphagia (B *Right*) Partial emptying of the hypopharynx. The right lateral pharyngeal pouch and the valleculae are filled with barium. The two small pouches on the left side of the hypopharynx are not visible at this time.

FIG 15 Note the bilateral pouches high up in the hypopharynx. The pouch on the right side is apparently associated with a secondary diverticulum originating from it.



disturbances of deglutition. The disease usually occurs in early life—as a rule after puberty. There is a tendency to spontaneous remissions. The fact that the course of the disease may be altered during pregnancy or because of changes of thyroid function suggests that the disease is fundamentally of an endocrine or metabolic nature.

Examination at autopsy of those cases dying of the disease shows no structural changes in the nerves or the muscles which would give a clue to the underlying pathology. The only suggestive finding is that of hyperplasia or tumor of the thymus. The disease has been compared with curare poisoning. In both conditions electromyo-

graphic studies show a block to the normal neuromuscular function. The motor fibers act normally. The response of muscle to direct stimulation is also normal. The difficulty therefore appears to lie in the transmitting machinery from the motor end-plates to the muscle. Both conditions also respond to anticholinesterase drugs such as eserine and neostigmine. The administration of curare makes the patient with myasthenia gravis much worse. McEachern²⁸ believes that the disease may be due to a deficient synthesis or liberation of acetylcholine at the motor end-plates. The only evidence that the thymus may have a curarelike function rests on the clinical evidence of improvement after thymectomy.

Radiologically, evidence of involvement of the pharyngeal muscles may be shown by disturbance in the activity of the hypopharynx with abnormal delay in the empty-

ing of the valleculae and the pyriform sinuses.

Abnormally prolonged retention of barium within the recesses of the hypopharynx in myasthenia gravis is illustrated by the following case.

D. G., female, aged 19, was admitted to the hospital with a history of progressive exophthalmos and generalized myopathy. She had noticed increasing prominence of her eyes and excessive tearing. This condition became progressively worse until she developed amblyopia. She also noticed the gradual onset of weakness in both arms and legs and later of her neck. The weakness became so marked that she was unable to hold things in her hands and her legs were no longer able to support her in any attempt to stand so that she kept falling. At this time she noticed marked weakness of her jaw muscles which prevented her from chewing adequately. She had episodes of respiratory embarrassment, accompanied by a feeling of constriction in

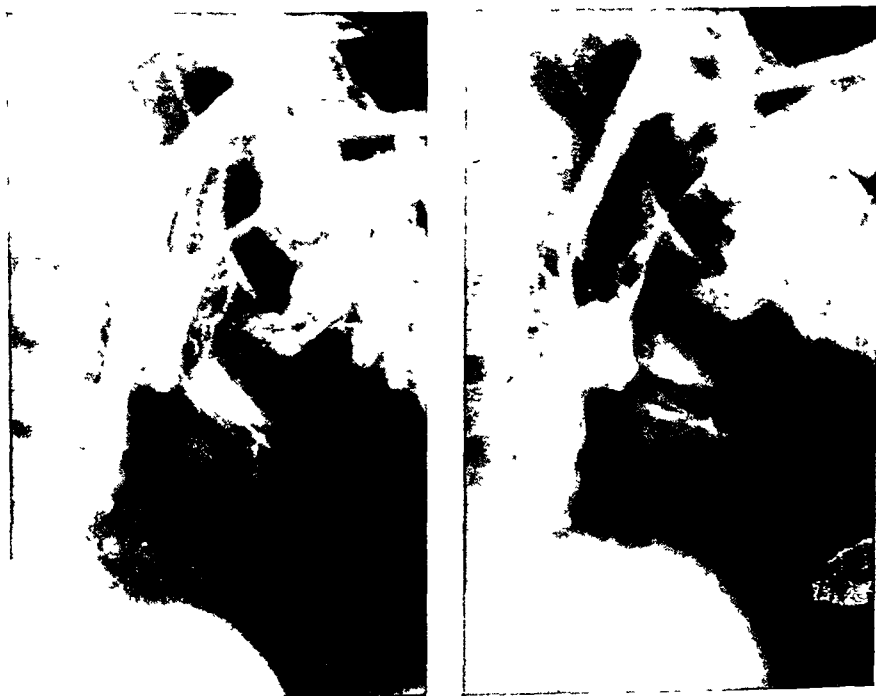


FIG. 16 The hypopharynx in myasthenia gravis (A, *Left*) Appearance of the hypopharynx 3 minutes after the administration of the barium suspension (B, *Right*) Appearance 5 minutes later (8 minutes after the original administration of the barium suspension). Note that the entire hypopharynx is filled with barium corroborating the clinical diagnosis of myasthenia gravis. After the intramuscular administration of prostigmin re-examination showed that the hypopharynx emptied itself promptly of the barium suspension without any trace remaining.

the lower part of her neck or high in the chest which was partially relieved by sitting up. She also complained of difficulty in swallowing. Fluids would regurgitate unaccompanied or preceded by nausea so that tube feeding was

attempted in an effort to improve her state of nutrition and hydration. She salivated profusely at times and her inability to swallow the material gave rise to choking, dyspnea and cyanosis. Physical examination revealed a pal-

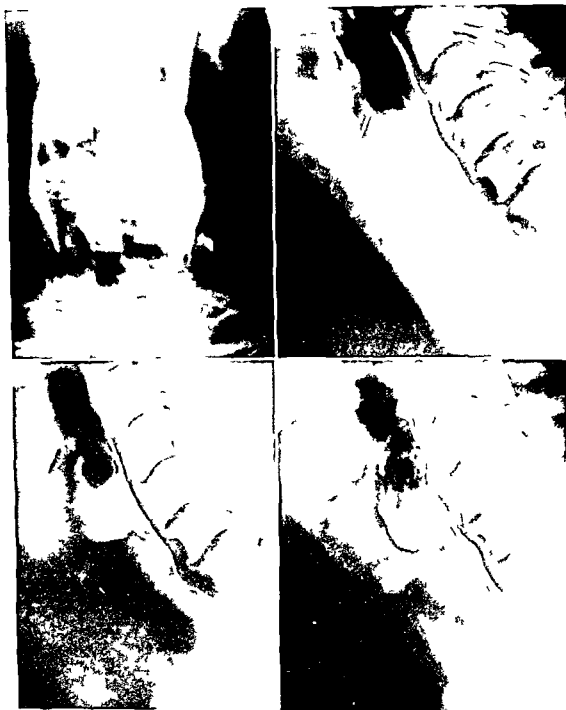


FIG 17 Pharyngeal dysphagia in bulbar palsy (A *Top left*) Postero anterior position. The appearance of the hypopharynx in a patient with bulbar palsy immediately after the ingestion of the barium suspension (B, *Top right*) Appearance in the left lateral position (C *Bottom left*) Appearance 15 minutes later (D *Bottom right*) Appearance one hour after Figure 17 A. Note the abnormal delay in the emptying of the hypopharynx.

pable, enlarged and slightly nodular thyroid and a persistent tachycardia of 90 or more. There was marked atrophy of all skeletal muscles.

Neurologic examination disclosed marked generalized weakness of practically all muscles of the body. She was unable to close her eyelids completely, to swallow fluids or to sit up, stand or walk by herself. She had to be lifted bodily into a wheel chair from her bed.

The clinical diagnosis was hyperthyroidism and myasthenia gravis.

The patient showed clinical improvement after the intramuscular injection of neostigmine methylsulfate. Following treatment with this drug she was able to close her eyelids completely. Her other muscular responses were improved. She continued to show improvement on a daily dose of 120 mg of neostigmine. Particularly striking was her ability to eat without gagging and to speak normally.

The evidence on radiographic examination of the hypopharynx is shown in Figures 16 A and B. Figure 16 A shows the distention of the hypopharynx 3 minutes after swallowing the barium suspension. Figure 16 B, taken 5 minutes later (8 minutes after the original administration), shows the persistent retention of the barium in the hypopharynx. The retention is not only in the recesses, but also the entire hypopharynx is filled with the suspension. Then the patient was given an intramuscular injection of 1 cc of neostigmine methylsulfate. After about 15 minutes she complained of a slight feeling of warmth. She began to move her head, neck and arms spontaneously, and her speech became much clearer. Then the hypopharynx was re-examined. The barium suspension passed through the hypopharynx promptly without any trace of retention.

The radiographic evidence confirmed not only the therapeutic effectiveness of the medication but also the diagnosis of myasthenia gravis.

BULBAR PALSY AND DYSPHAGIA

Paralysis of the gullet, as a cause of dysphagia, was recognized by Galen,²⁹ who called the condition *imbecillitas gulae* in order to distinguish it from dysphagia due to organic disease.

Wepfer, in 1757,³⁰ recorded several cases of dysphagia which developed after attacks

of apoplexy so severe that the difficulty in deglutition was the immediate cause of death. Dysphagia may be caused by various diseases affecting the central nervous system, such as hemorrhage into the pons or the medulla or tumors in these regions, bulbar paralysis, multiple sclerosis or cerebral atrophy in general paralysis of the insane.

Dysphagia as a complication of bulbar palsy in poliomyelitis is well recognized.

The disturbance in the motor function of the hypopharynx present in bulbar palsy may be demonstrable roentgenologically.

G. D., female, aged 68, was admitted to the hospital complaining of dysphagia, headache and slight fever of 10 days' duration. The onset of the dysphagia was sudden and was accompanied by nasal regurgitation. Because of the elevated temperature (100.3° F) on the third day of admission, a spinal fluid tap was done which revealed signs of inflammation of the central nervous system. A diagnosis of a mild bulbar form of poliomyelitis was made. The possibility of a neoplasm of the hypopharynx was considered at that time. However, endoscopic examination failed to reveal any sign of tumor. During the period of clinical activity the patient was observed while attempting to swallow fluids. She experienced extreme difficulty in doing so in spite of repeated efforts. Some of the fluid escaped by way of the mouth and the nose. The patient made a gradual clinical improvement, although at the time of her discharge from the hospital there was still some residual evidence of dysphagia. The clinical observations regarding the dysphagia were confirmed by the radiographic examination of the hypopharynx. Figure 17 A (postero-anterior view) shows the appearance immediately after the swallowing of barium. Figure 17 B shows the appearance in the left lateral position. Figure 17 C shows the appearance 15 minutes later. Figure 17 D shows the appearance one hour after the original ingestion of the barium suspension. Note that even at the end of one hour the hypopharynx contains almost as much barium as was present originally. There is no evidence of any organic abnormality, the condition being due entirely to a disturbance in the swallowing mechanism, evidently the result of bulbar palsy.

REFERENCES

- 1 Mosher, H P X ray study of movements of the tongue epiglottis and hyoid bone in swallowing, etc, *Laryngoscope* 37 235, 1927
- 2 Killian G Über den Mund der Speiseröhre *Ztschr f Ohrenheilkunde* 55 1, 1908
- 3 Blankenstein Paul *Dysphagia Hysterica*, dissertation, Bonn 1893
- 4 Paterson, D R The direct examination of oesophagus and upper air passages, *Brit M J* 2 353 1906
- 5 Mosher, H P Webs and pouches of the esophagus, their diagnosis and treatment *Surg Gynec & Obst* 25 175, 1917
- 6 Kelly A Brown Spasm at the entrance to the oesophagus, *J Laryng & Otol* 34 285, 1919
- 7 Paterson, D R A clinical type of dysphagia, *J Laryng & Otol* 34 289, 1919
- 8 Suzman, M M Syndrome of anemia, glossitis, and dysphagia, *Arch Int Med* 51 1 1933
- 9 Hoover, W B The syndrome of anemia, glossitis and dysphagia, *New England J Med* 213 394, 1935
- 10 McGee, L C and Goodwin T M The syndrome of dysphagia and anemia *Ann Int Med* 11 1498, 1938
- 11 Ahlbom H E Simple achlorhydric anaemia, Plummer Vinson syndrome and carcinoma of the mouth pharynx and esophagus in women, *Brit M J* 2 331, 1936
- 12 Simpson, R R Anaemia with dysphagia a precancerous condition? *J Laryng & Otol* 54 738, 1939
- 13 Waldstrom, J and Kjellberg, S R The roentgenologic diagnosis of sideropenic dysphagia, *Acta radiol* 20 618 1939
- 14 Vert, H R Diagnosis of myasthenia gravis in patients with dysphagia, *J A M A* 134 987 1947
- 15 Wenglowski Romuald Über die Halsfisteln und Cysten *Arch klin Chir* 98 151 1912
- 16 Bailey, Hamilton The clinical aspects of branchial fistulae, *Brit J Surg* 21 173, 1933
- 17 Sommer, G N G, Jr, Conley, J J, and Dunlay, H J Cervical lesions of branchial origin *Am J Surg* 61 266, 1943
- 18 Douglas, John Branchiogenic cyst with sinus leading to pharynx, *Ann Surg* 67 240, 1918
- 19 Bailey, Hamilton The clinical aspects of branchial cysts, *Brit J Surg* 10 565, 1922 23
- 20 Carp Louis Branchial fistula—its clinical relation to irritation of the vagus, *Surg, Gynec & Obst* 42 772, 1926
- 21 Shedden William M Branchial cysts and fistulae *New England J Med* 205 800, 1931
- 22 Meyer, Herbert Willy Congenital cysts and fistulas of the neck, *Ann Surg* 95 1, 1932, 95 226, 1932
- 23 Ladd W E, and Gross, R E Congenital branchiogenic anomalies A report of 82 cases *Am J Surg* 39 234, 1938
- 24 Hoover, Walter B Clinical conditions resulting from anomalies or maldevelopment of the branchial arches and clefts *Ann Otol, Rhin & Laryng* 50 854, 1941
- 25 Godlee, R J, and Bucknall, T R H A pharyngeal pouch of large size removed by operation *Lancet* 1 1387, 1901
- 26 Raven, R W Pouches of the pharynx and oesophagus with special reference to the embryological and morphological aspects *Brit J Surg* 21 235, 1939
- 27 Buckstein Jacob and Reich, Stanley Lateral pharyngeal diverticula as a cause of dysphagia *J A M A* 144 1154, 1950
- 28 McEachern Donald Diseases and disorders of muscle function, *Bull New York Acad Med* 27 3 1951
- 29 Galen *De locis affectis lib II, cap v*, 1548
- 30 Wepfer *Historia Apoplect Venetus*, 1757, p 376

The Normal Esophagus

ANATOMIC CONSIDERATIONS

The esophagus is about 25 cm long. It extends from the lower end of the pharynx to the cardiac end of the stomach, passing through the superior and posterior mediastinum and piercing the diaphragm at the level of the tenth thoracic vertebra.

Liquids on entering the esophagus may reach the lower end in one tenth of a second. Solid or semisolid food is forced down by peristaltic activity, as originally described by Cannon and Moser¹ in 1898. The circular muscle is constricted from above downward by the peristaltic wave, which takes about 5 or 6 seconds to reach the cardia. For this purpose of rapid contraction the muscle in the upper part of the esophagus is striated. This type of muscle fiber gradually disappears until, in the lower portion, only smooth muscle is found. There appears to be no pause in the downward peristaltic movement, about 6 seconds being taken for the passage of the wave until it reaches the stomach.

The factor of gravity in the swallowing mechanism was described by Schieber,² who examined patients during the act of swallowing a bismuth suspension while standing on their heads, his purpose being to determine the role of gravity in the act of deglutition. He stated that under these circumstances the act of swallowing was insufficient to overcome the factor of gravity, and the bismuth suspension could not be forced beyond the proximal area of the esophagus.

That the act of swallowing may be successfully accomplished with the patient standing on his head was shown by Thomas.³ The patient was observed fluoroscopically,

and the rate of progress of the opaque cream through the esophagus into the stomach appeared to be essentially like that taking place with the patient standing on his feet. A pictorial presentation of the patient standing on his head and of the roentgenogram which was taken accompany the article. The entire esophagus is well outlined and the opaque material may be noted making its exit through the cardia and entering the stomach. The importance of the factor of gravity, however, is quite obvious and in the erect position aids materially in the rapid descent of the swallowed substance through the pars cardia into the stomach.

Although the general direction of the esophagus is in a vertical line, there are two deviations in its course: one is near the beginning, where it deviates to the left toward the root of the neck, and the other is also to the left as it makes its way through the esophageal hiatus.

Areas of normal physiologic constriction are present:

- 1 At the junction of the pharynx and the esophagus, at the level of the cricoid cartilage.

- 2 At the crossing of the aorta.

- 3 In the region where the esophagus passes through the hiatus of the diaphragm. The degree of constriction here will depend upon the physiologic state of the diaphragm. The narrowing will be less marked if the muscle fibers of the diaphragm are relaxed.

At times, a fourth area of constriction produced by the left bronchus may be encountered.

Other areas of the esophagus may appear constricted because of its somewhat

twisted course Pratte¹ explained in this manner an apparent constriction in the lower end of the esophagus a few centimeters above the diaphragm. Under such circumstances the esophagoscopist may note an abrupt closure of the lumen, which he may misinterpret as being due to an actual area of constriction.

Below the impression on the esophagus produced by the aortic arch sometimes one may note the effect upon it of the pressure produced by the left bronchus. In order to determine the relation of the right pulmonary artery to the left bronchus and the esophagus, Evans made a careful dissection of the posterior mediastinum in three cadavers. He found that neither the pulmonary artery stem nor its branches were in direct apposition to the esophagus. The pulmonary artery stem divided in front of the left bronchus. The right pulmonary artery was separated from the esophagus by a space filled with connective tissue and lymph nodes. He also impregnated the dissected specimens with barium and made roentgenograms showing that the impression on the esophagus below the aortic arch was produced by the left bronchus. However, this impression by the left bronchus may be influenced secondarily by the pressure of an abnormal right pulmonary artery.

At times, it may be possible to see three backward curves from above downward: (1) the aortic impression, (2) the left bronchus impression and (3) the left auricular impression. In some cases there is a combination of the impressions produced by the aortic arch and the left bronchus, so that there is a single curve above the left auricular curve. The impression produced by the pulmonary artery in disease results primarily from the fact that the distended pulmonary stem causes pressure of the left bronchus against the esophagus. Such exaggeration of the normal impression produced by the pulmonary artery may occur in congenital heart disease, mitral stenosis, emphysema and aneurysm of the pulmonary artery.

The cardiac sphincter depends on its intrinsic musculature and is functionally independent of the musculature of the diaphragm, although in some cases the latter may be an accessory aid in augmenting the sphincteric activity of the cardiac end of the esophagus. This independence of the cardiac sphincter is shown by the fact that the sphincteric region of the esophagus may be located above the opening of the diaphragm, and also by the fact that in herniation of the stomach the cardiac sphincter is definitely independent of the diaphragm.

The folds of the esophagus are longitudinal and, for the most part, parallel. They vary from two to five in number and may show overlapping of those on the anterior and the posterior walls. In rare cases they may be undulating; occasionally they are decussated. Normally, the folds may be noted through the extreme cardiac end of the esophagus, and their demonstration in this region may assume considerable diagnostic importance in the differentiation of a benign from an infiltrating lesion.

TECHNIC OF ROENTGEN EXAMINATION

As a precaution before examining the esophagus it is essential that the patient reporting in the morning shall have had no food since the preceding evening because food particles retained in the esophagus may produce translucent areas within the barium suspension. These may be misinterpreted as being the result of intrinsic disease within the esophagus itself. Such misinterpretation is particularly likely to occur in patients with cardiospasm. Food retained at the extreme cardiac end may distort the contour and produce an irregularity simulating that of actual new growth.

In some cases direct fluoroscopic observation of the esophageal area may yield valuable information prior to the administration of any opaque substance for its visualization. This is particularly true in some cases of cardiospasm, in which the hugely dilated esophagus may be made

visible by the retained fluid within it. As a rule, however, the administration of opaque material for the delineation of the esophagus is essential.

In those cases in which the esophagus is examined in routine fashion as part of a study of the stomach (the details of which will be discussed later), no special mixture is employed other than that for the stomach itself. When a special study of the esophagus is to be made because a lesion is suspected, a thicker mixture of barium is needed. The usual barium-water mixture for the study of the stomach passes through the esophagus with considerable rapidity, and a detailed, prolonged visualization of an organically involved area may be difficult or impossible. However, a thicker mixture will pass through the esophagus much more slowly. Prolonged visualization of the esophagus is thereby aided, and films may be taken which will more readily demonstrate any lesion that may be present.

The mixture employed at Bellevue Hospital is made up of barium sulfate and acacia, a sufficient amount of the acacia being added to the barium sulfate to thicken it so that it barely runs off the spoon. This should be stirred thoroughly until completely homogeneous. Small, discrete masses of barium may obscure the picture and give a misleading appearance to the roentgenogram. A mixture of this consistency will stick to the esophagus long enough for proper visualization and only a very small quantity is needed. As little as one teaspoonful of this mixture will clearly delineate an infiltrating new growth. Moreover, when the lesion is of a markedly obstructive nature, only a small amount of the barium should be administered since, otherwise, there may be a reflux of the suspension into the trachea and the bronchi by way of the pharynx. In some cases, when the obstruction is extreme, as thin a mixture of barium as possible is advisable in order to prevent regurgitation.

The nature of the mixture and the position of the patient during fluoroscopy and

roentgenography are ordinarily essentially identical regardless of the character of the organic lesion of the esophagus which is suspected. In special cases, such as, for example, in the roentgen demonstration of esophageal varices, further details in the technical management are essential. Such variations in technic, however, will be discussed in connection with the particular problem involved.

In the roentgen examination of the esophagus, the position of the patient is of considerable importance. At the very outset the patient is placed behind the fluoroscopic screen in the erect position. However, for visualization of the esophagus this position is ordinarily unsatisfactory since the esophagus, for the most part, is obscured by the shadow of the heart. Therefore, in order to isolate the esophagus and bring it into clear relief, the patient is turned obliquely so that his right side is against the fluoroscopic screen, a position commonly referred to as the "first oblique." Not only is this position ideal for the study of the esophagus when intrinsic lesions of this region are suspected but it is also important in the study of the stomach as well, as will be discussed more fully later. As the barium is swallowed, each part of the esophagus is closely scrutinized down to the cardiac end. As in fluoroscopy in general, the screen should be focused as much as possible so as to prevent the escape of unnecessary radiation, as well as for increased clearness of detail.

Following observation in the first oblique position, the patient should be turned through every angle of obliquity, since lesions of the esophagus not clearly demonstrable at one angle may thereby be made visible at another.

Films taken after completion of the fluoroscopic study can be taken in both the erect and the prone positions. The prone position, however, is ordinarily preferable, the patient being turned through that degree of obliquity which preliminary fluoroscopic observation has shown to be best.

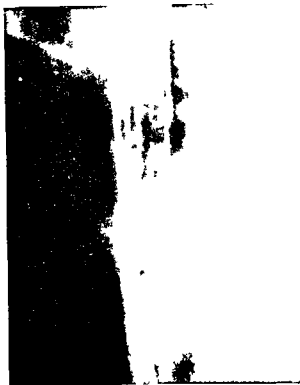


FIG 18 (A *Left*) Normal esophagus, showing from above downward (1) the aortic impression (2) the left bronchus impression and (3) the left auricular impression (B, *Right*) Normal mucosal folds of the esophagus



FIG 19 (A *Left*) Air bubbles in the esophagus (B, *Right*) Same case as is shown in A Note the complete disappearance of the air bubbles at this time

suited for the clear demonstration of the lesion

Spot film technic may enhance the value of the radiographic exposure by fixing on

the film the exact area of the esophagus under suspicion as determined by fluoroscopic examination

Figure 18 A shows the normal esophagus



FIG 20 Normal physiologic behavior of the cardiac end of the esophagus independent of the diaphragm. Note that the cardiac end joins a herniated portion of the stomach well above the diaphragm. (A, *Left*) The esophagogastric junction is wide open at this time. (B, *Center*) Shows a moderate degree of contraction at this junction. (C, *Right*) Shows an almost complete contraction at this junction.

exhibiting, from above down. (1) the aortic impression, (2) the left bronchus impression and (3) the left auricular impression.

Figure 18 B shows the presence of the longitudinal folds of the esophagus.

Small globules of swallowed air may be trapped within the esophagus and may simulate polypoid lesions. However, the onward passage of these globules may be noted during fluoroscopy, and repeated roentgenograms will show the transient character of the alterations.

Figure 19 A shows small translucent globules within the esophagus.

Figure 19 B shows the disappearance of the globules, indicating their transient character, the result of swallowed air.

That the closure mechanism at the cardiac end of the esophagus is independent of the hiatal opening in the diaphragm is illustrated by Figure 20. In this case the distal end of the esophagus joins a herniated portion of the stomach well above the diaphragm. In Figure 20 A the esophagogastric junction is wide open. In Figure 20 B there is evidence of a closure mechanism in this region. A further advance in the degree of contraction at this junction may be noted in Figure 20 C. Thus, one may note the functional behavior of the cardiac end of the esophagus, completely independent of any diaphragmatic factor. However, the

contraction of the diaphragm has an accessory role in aiding in the closure of the distal end of the esophagus when it passes through the hiatal opening. If the barium-filled esophagus is observed carefully it will be noted frequently that during deep inspiration there is temporary delay in the emptying of the distal portion as well as an ampullary dilatation of this segment. Thus, while the closure mechanism of the cardiac sphincter is essentially independent, the contraction of the diaphragm during inspiration may be a contributing factor.

An important anatomic feature at the distal end of the esophagus is the transient dilatation which sometimes may simulate a small gastric hernia. It has been described variously as the "epicardia" (Schreiber), the "epiphrenic bell" (Anders), the "cardiac antrum" (Luschka) or more popularly as the "phrenic ampulla" (Hasse and Strecker). The mechanics of its formation probably depend on the phreno-esophageal membrane running from its attachment at the diaphragm to a variable point on the esophagus that is a short distance above the cardiac end.⁶

The segment distal to the constriction produced by the insertion of the phreno-esophageal membrane may then balloon out, at times, particularly during inspiration when the descent of the diaphragm causes

further physiologic occlusion of the cardiac end

Therefore the position as well as the configuration of the phrenic ampulla apparently depends on the following factors:

1 The exact anatomic position of the insertion of the phrenoesophageal membrane

2 The anatomic position of the distal end of the esophagus

3 The length of the segment between the insertion of the membrane and the cardiac end

4 The anatomic relation of the hiatus of the diaphragm to the segment of the esophagus running through it

5 The moment of the respiratory phase the ampullary dilatation being most marked in deep inspiration and as a rule disappearing in expiration

The appearance and the behavior of the phrenic ampulla may be noted with particular clarity under fluoroscopic observation. When the esophagus is filled with barium, and the patient is told to take a deep breath, the distal end will balloon out only to disappear with expiration. The phrenic ampulla may be globular or ovoid depending not

only on those factors which have been noted already but also on the degree of the momentary distention with barium. Both fluoroscopically and roentgenologically the configuration of the ampulla may vary from moment to moment in the same individual. Indentation of the ampulla may be produced by the concentric hiatus of the diaphragm.

In some cases it is quite possible that the phrenic ampulla may occur independently of a phrenoesophageal membrane. It may be the result of a distordence in the swallowing mechanism so that the cardiac end does not open synchronously with the advancing peristaltic wave. The swallowed barium meeting with resistance, then may cause a transient dilatation of the distal portion. Similarly, the occlusion of the distal end of the esophagus produced by the diaphragm in inspiration also may cause a sufficient arrest of the barium to lead to dilatation without the necessity of a membrane. A concomitant contraction at the insertion of the phrenoesophageal membrane.

An ampullary dilatation of the distal end of the esophagus (phrenic ampulla) illustrated by Figure 21. Note that in Figure



FIG. 21 (A, Left) Note the phrenic ampulla at the distal end of the esophagus (B, Right) The phrenic ampulla has disappeared and has become incorporated with the rest of the esophagus. The fact clearly differentiates it from a constriction of the stomach.



FIG 22 (A, *Left*) Ampullary dilatation of the distal end of the esophagus (phrenic ampulla) (B, *Left center*) Note that at this stage there is considerable reduction in the size of the phrenic ampulla, although all of the technical factors were identical Note also the alteration in the contour of the ampulla (C, *Right center*) Still further reduction in the size of the phrenic ampulla Note also the change in shape (D, *Right*) Disappearance of the phrenic ampulla At autopsy the esophagus appeared to be entirely normal The changes noted were obviously of a functional nature

21 A there is a rounded ampullary dilatation of the distal end of the esophagus In Figure 21 B the esophagus appears quite normal, and the ampulla has disappeared Note that the position of the patient is identical in both observations There were no symptoms attributable to this condition Fluoroscopic examination is paramount in the differentiation of a phrenic ampulla from a small hernia of the stomach through the esophageal hiatus One may note that the ampullary dilatation is actually part of the esophageal structure itself and is of a transient nature, depending upon the functional behavior of the esophagus Under fluoroscopic control the tendency to develop a phrenic ampulla may be exaggerated by having the patient take and hold a deep breath In such cases the constriction of the esophageal hiatus upon the distal end of the esophagus is apparently an important factor in causing the appearance of a phrenic ampulla A small hernia of the stomach protruding through the esophageal hiatus produces a saccular area independent of the esophagus itself, which will not disappear because of functional changes in the behavior of the esophagus

The functional behavior of the distal portion of the esophagus with the formation of a phrenic ampulla is illustrated particularly in the case of C S, male aged 83 This patient died of a carcinoma of the stomach which had been diagnosed preoperatively In addition, radiographic examination of the esophagus showed the variations in the configuration of the distal portion noted in Figure 22 An autopsy was performed, and the diagnosis of carcinoma of the stomach was confirmed The esophagus was free of any organic abnormality Therefore, the changes noted on radiographic examination of the esophagus were of a functional nature In Figure 22 A there is a well-formed phrenic ampulla which simulates a small hernia of the stomach through the esophageal hiatus However, note that in Figure 22 B there is a marked diminution in the size of the phrenic ampulla, although the possibility still might be entertained that it represented a small hiatal hernia Also, note the change in the contour of the phrenic ampulla In Figure 22 C there is further diminution in the size of the ampulla, it now appears to be more closely linked with the rest of the esophagus At

this time the ampulla is quite well rounded. Finally, in Figure 22 D the ampullary dilatation has disappeared completely and the distal cardiac end now quite obviously appears to be continuous with the rest of the esophageal contour. It is quite clear then that the dilatation at the distal end of the esophagus is actually a phrenic ampulla and not the result of organic pathology in the esophagus or of a small hiatal hernia. Particularly significant is the fact that these observations were checked by autopsy. The transient nature of such ampullary dilatations of the distal end of the esophagus may be noted best by observation under fluoroscopic control, which will clearly aid in the differentiation of this condition from an actual herniation of the stomach.

THE ESOPHAGUS IN INFANCY

A study of the roentgen appearance of the esophagus in normal infants was made at Bellevue Hospital in 1944.⁷ The study was based on the examination of 32 infants, ranging in age from 3 weeks to 22 months, all of whom were free of gastro intestinal symptoms. The observations showed that the esophagus in infants is markedly dilatable and during the course of the feeding of the barium acacia mixture the lumen was often as wide or even wider than the vertebral column approximating the diameter of a normal adult. That the lumen of the esophagus is relatively larger in infants than the lumen of adults also has been shown by Melencini, Roca and Banzis.⁸ Such variation in the potential distensibility of the esophagus in infancy within the range of the normal must be considered before one commits oneself to a diagnosis of cardiospasm. Moreover regurgitation of the barium from the stomach into the esophagus may give rise to a false conception of

abnormal retention due to a pathologic condition of the cardiac sphincter, a point to be determined only by fluoroscopic observation.

In some cases vomiting in infancy may be caused by abnormal relaxation of the cardio-esophageal junction.⁹ In this condition the barium in the stomach may be noted welling up into the esophagus without hindrance in retrograde fashion. The condition appears to be a temporary disturbance of the neuromuscular factors controlling the behavior of the cardiac end of the esophagus.

Figure 23 shows the appearance of the normal esophagus of an infant.



FIG. 23 Normal esophagus in an infant.

REFERENCES

- 1 Cannon, W B, and Moser, A The movements of the food in the oesophagus, *Am J Physiol* 1 435, 1898
- 2 Schrieber, J Über den bewegenden Einfluss der Schwerkraft beim Trinken in Aufrechter und Kopfstellung, *Arch Verdauungskr* 21 1, 1915
- 3 Thomas, A R A note on deglutition, *Brit J Radiol* 15 209, 1942
- 4 Pratje, A Form und Lage der Speiserohre des lebenden Menschen, ein Beitrag zur Topographie des Mediastinum, *Ztschr f d ges Anat* 81 269, 1926
- 5 Evans, William The Course of the Oesophagus in Health and Disease of the Heart and Great Vessels, No 208, Medical Research Council, 1936
- 6 Lerche, William The Esophagus and Pharynx in Action, chap 5, Springfield, Thomas, 1950
- 7 Bakwin, H, Galenson, E, and LeVine, B E Roentgenographic appearance of the esophagus in normal infants, *Am J Dis Child* 68 243, 1944
- 8 Melenchini, M, Roca, J, y Banzas, T Aspecto radiológica del esófago en el lactante, *Prensa med argent* 35 1698, 1948
- 9 Berenberg, W, Neuhauser, E B D Cardio-esophageal relaxation (Chalasia) as a cause of vomiting in infants, *Pediatrics* 5 414, 1950
- 10 Palmer, Eddy D The Esophagus and Its Diseases, New York, Hoeber, 1952

Congenital Anomalies of the Esophagus

ANOMALIES OF POSITION

Displacement Displacement of the esophagus may result from congenital anomalies in the position of the right or the left subclavian artery.

Davis Bayford¹ in 1794 described as dysphagia lusoria the dysphagia produced by the pressure on the esophagus of an abnormally situated right subclavian artery. In Bayford's case, the right subclavian artery crossed between the esophagus and the trachea, causing constriction of the esophagus with difficulty in swallowing and eventual death from inanition.

A reproduction of the anatomic findings in Bayford's case is to be found in Quinn's famous atlas of lithographic drawings of the anatomy of the arteries.

The relation of the right subclavian artery to the esophagus and the trachea is shown in the classical survey made by Holzäpfel in 1899² of all the cases available in the literature in addition to four cases of his own. In 6 cases or 5 per cent the artery was in front of the trachea. In 20 cases, or 15 per cent the vessel was between the trachea and the esophagus. In 107 cases or 80 per cent, the artery was present behind the esophagus. The vessel was fixed by connective tissue to the vertebral column and crossed it in an area between the fourth thoracic and the sixth cervical vertebra. In 51 cases a study of the caliber of the vessel was made. In 18 cases the caliber of the anomalous right subclavian artery was approximately that of the left subclavian artery. In 33 of these cases the lumen of the vessel at its source of origin was larger than the left subclavian artery. Compression of the posterior wall of the esophagus may

depend at least in part, on the prominence of the blood vessel.

This assumption received further support from the observation made by Harvey³ who found 2 cases of an anomalous right subclavian artery in 237 cadavers, or 0.8 per cent. In one of these cases the vessel was considerably dilated up to the point where it emerged behind the esophagus. Probably only an abnormally dilated vessel is capable of producing roentgenologically recognizable evidence in the examination of the barium filled esophagus. Certainly the anomalous vessel would have to be of rather large caliber in order to produce sufficient constriction of the esophagus to cause dysphagia.

In his survey Holzäpfel also included an interesting reference to the existence of an anomalous right subclavian artery in lower animals. One case of an anomalous right subclavian artery in one of six hedgehogs that he had studied, was described by Meckel in 1810. The vessel originated on the left side and then passed behind the esophagus to the right side. Another case, in a rabbit was described by W. Ramsay Smith in 1891. In this case also the right subclavian artery originated on the left side crossed behind the trachea and the esophagus and then continued its usual course.

A remarkable case was described by Kirby in 1818 in which the anomalous position of the right subclavian artery because of pressure contributed to the death of the patient. The swallowed food had been arrested in the upper portion of the esophagus. Autopsy revealed three large morsels of food impacted in the esophagus. The more distal one contained a piece of bone $\frac{1}{2}$ inch long which perforated the

right subclavian artery, which, "contrary to its usual course and origin, lay in this situation as it passed from the left of the arch of the aorta, where it arose, towards the right shoulder"

Pressure from the aberrant subclavian vessel may well have been sufficient to arrest the further progress of the large morsel of food at the particular level where the bone was able to perforate it with fatal hemorrhage resulting

Clinical evidence of obstruction by an anomalous right subclavian artery is also suggested by Batten's⁶ experience. A boy, aged 2 years and 10 months, was severely choked while eating an apple. He recovered only to undergo a similar experience a few months later while eating dinner, this time ending fatally. Autopsy revealed impaction of a mass of food, apparently sausage, above the level of a right subclavian artery which arose from the descending part of the arch of the aorta and crossed obliquely behind the esophagus to its usual position at the root of the neck.

An anomalous right subclavian artery frequently may be associated with congenital anomalies of the heart. Brean and Neuhauser⁷ found the combination of a patent ductus arteriosus and an aberrant right subclavian artery with sufficient frequency to suggest that the relationship was probably more than fortuitous.

In the case reported by Shellshear and Anderson⁸ a newborn male child died with clinical evidence of esophageal atresia. Autopsy revealed an incomplete atresia of the esophagus at the exact level where an anomalous right subclavian artery compressed it posteriorly. I have been unable to find any other report of a similar association. Probably it must be considered as a strange coincidence.

Roentgen Diagnosis Apparently the first roentgen demonstration of the deformity of the esophagus produced by an anomalous right subclavian artery and the aortic diverticulum was by Kommerell.⁹

The roentgen diagnosis of an aberrant

right subclavian artery depends upon the following main features

1 The pressure upon the left lateral border of the esophagus produced by the aortic arch in the postero-anterior position

2 The concave defect upon the right lateral border of the esophagus at a level higher than the indentation produced by the left aortic arch. The relative position of these two indentations may be particularly well noted in the right oblique position. Since the anomalous right subclavian artery has its origin on the left side and passes obliquely upward on its way to the right, therefore the pressure defect upon the esophagus when it runs posterior to it will be at a higher level than the defect produced by the left aortic arch. The characteristic appearance then in the right oblique position is that of an indentation upon the anterior wall produced by the aortic arch and a smooth pressure defect on the posterior wall of the esophagus at a slightly higher level.

3 Because of the anatomic direction of the anomalous artery, passing as it does from left to right in an upward direction, roentgen examination, particularly in the left anterior oblique position, will show an oblique filling defect of the esophagus.^{10,12}

Neuhauser has emphasized the importance in the recognition of an anomalous right subclavian artery of the oblique defect produced by the vessel on the posterior aspect of the barium-filled esophagus, as it passes upward from left to right, frequently at a level just above the indentation produced by the normally placed left aortic arch. This oblique bandlike defect occasionally may be noted in the anteroposterior position, particularly if the compressed portion of the esophagus contains only a small amount of barium. Overdistention of the esophagus with barium may obscure the imprint upon it produced by the vessel. The oblique semitranslucent band may be seen in the left anterior oblique position. This band may also be seen in the right anterior oblique position, as in the next case.

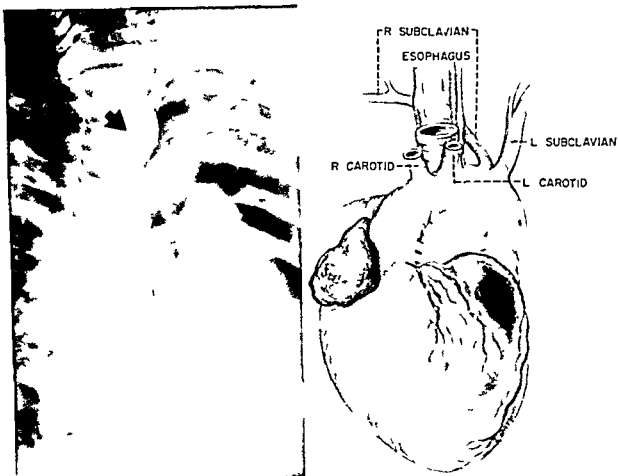


FIG. 24 (A *Left*) Anomalous right subclavian artery. Examination of the esophagus in the right oblique position shows the following (1) Normal left sided position of the aortic arch (2) narrow semitranslucent band extending in an oblique upward direction toward the right side (3) forward displacement of this segment of the esophagus (4) during fluoro copy a delay was noted in the passage of the barium at about the level of the semitranslucent band (B *Right*) Reproduction of the anatomic findings as found at an autopsy showing the retro esophageal position of the anomalous right subclavian artery

When the retro esophageal concave defect is unusually wide one may assume that it is due not only to the pressure of the right subclavian artery but also in varying degree to the associated compression produced by the aortic diverticulum from which the vessel has its anomalous origin. Only such an assumption can explain logically a defect too wide to be accounted for by the mere pressure of the subclavian artery itself unless it were the site of an aneurysmal dilatation.

In addition to the localized pressure on cavity the segment of the esophagus in this

region may show constriction of the lumen. Fluoroscopy may also demonstrate that the extra esophageal mass is a pulsating structure. This feature may help to differentiate the condition from the pressure produced by a nonpulsating mass such as a node or a tumor.

The forward displacement of the esophagus produced by the combined pressure of the anomalous right subclavian artery and the aortic diverticulum may simulate the deformity produced by a right aortic arch. Differential diagnosis will then depend on the fact that in the postero anterior posi-

which had become dilated in part of its course

In some of the cases of right aortic arch displacement of the esophagus is produced by the pressure of a left subclavian artery originating on the right side and then passing behind the esophagus¹

Apparently the first roentgen description of a right sided position of the aortic arch is to be found in the paper by Mohr² Two cases are included with the actual roentgenograms showing the aortic arch on the right side Koentgen examination of the esophagus was not made in either case

Illustration of compression and displacement of the esophagus by a right aortic arch may be found in the contributions of Arkin³ Renander⁴ Fisher Hope and Kobin on⁵ Lowenack⁶ Biedermann⁷ Blackford Davenport and Bayley⁸ Bedford and Parkin on⁹ and Gray

The roentgen diagnosis of a right aortic arch is aided by the following features

1 Roentgenographic examination of the chest will show an absence of the aortic arch on the left side Instead a prominent shadow with systolic pulsations may be noted on the right side corresponding to the anatomic location of a right aortic arch

2 There may be evidence of a pressure defect with displacement of the esophagus to the left This pressure defect is smooth sharply defined and may cause considerable narrowing of the lumen at that level A rounded pulsation may be noted to the right of the displaced and compressed esophagus This evidence of pulsation caused by the right aortic arch may receive further confirmation from kymographic studies Angiocardiographic visualization of the aorta may delineate the exact anatomic position of the arch as well as the descending aorta

When the anomalous right aortic arch passes toward the left it will produce a smooth indentation upon the posterior wall of the esophagus with anterior displacement However it must be borne in mind

that such a deformity may also be produced by retroesophageal pressure of a diverticulum of the left aortic arch

When the concave pressure defect of the posterior wall of the esophagus is wide, probably it is due to a large right aortic arch or possibly a diverticulum of the left aortic arch A shallow defect is much more apt to be produced by an anomalous subclavian artery or a ductus arteriosus An aortic arch of smaller caliber when part of a double aortic arch may also produce a defect of less extent and depth than that produced by a single right aortic arch which carries the entire blood flow from the heart

3 When visualized in the roentgenogram the trachea may show displacement to the left by the right aortic arch The tracheogram produced by the instillation of a small amount of Lipiodol will accentuate more clearly such displacement

4 In some cases of a right aortic arch the ligamentum arteriosum may compress the pulmonary artery against the trachea and then pass posterior to the esophagus and join the aorta In this manner a complete vascular ring is formed which may seriously occlude the trachea and the esophagus As a result roentgen examination of the esophagus will show not only pressure of the right lateral border but also of the left lateral border Since the anterior portion of a double aortic arch may be much smaller than the right arch or may actually be obliterated to form a cordlike structure differential diagnosis is from the constriction caused by a ligamentum arteriosum may be difficult or impossible The subject of vascular ring formations will be discussed in greater detail later

5 Occasionally in the case of a right aortic arch the aorta descends entirely to the right of the esophagus and does not pass retroesophageally to emerge to the left of the spine Then the left subclavian artery originating from the right aortic arch may pass from right to left behind the esophagus The anomalous left subclavian artery then

tion the aortic arch is present on the left side

An example of the pressure defect produced upon the esophagus by an anomalous right subclavian artery is shown in the following completely documented case

J O, a male child, 2 years old, was admitted to Bellevue Hospital because of known congenital heart disease. The presence of an anomalous right subclavian artery was first recognized on fluoroscopic examination of the esophagus as part of an evaluation of the cardiac condition. The findings were best demonstrated in the right anterior oblique position. Examination in this position showed a striking tangential impingement on the barium-filled esophagus very high in the thorax with slight delay in the passage of the barium through this area.

The child's history and observation on the ward gave no evidence of difficulty in swallowing nor of increase of dyspnea during feeding. The clinical diagnosis of the underlying heart anomaly was a high ventricular septal defect. The child died as the result of progressive congestive cardiac failure.

Autopsy confirmed the clinical diagnosis of a ventricular septal defect. The diagnosis of an anomalous right subclavian artery was also confirmed. The artery was found to arise from the posterior aspect of the transverse arch of the aorta and passed from this area in an upward direction toward the right behind the esophagus into the right arm. There was no evidence of compression through the esophagus on the trachea.

The findings in the radiographic examination of the esophagus are shown in Figure 24 A. The child was in the right oblique position. The following features may be noted: (1) The aortic arch is on the left side. (2) There is a semitranslucent band across the upper portion of the esophagus extending in an upward direction toward the right. This band corresponds anatomically to the size of a blood-vessel structure. (3) There is a moderate degree of pressure on the posterior wall of the esophagus at the upper margin of this band, with forward displacement of this segment of the esophagus. Therefore, these findings justified the diagnosis of a blood vessel originating from the left-sided aortic arch and passing in an oblique upward direction toward the right side with forward displacement of the esophagus. (4) During fluoroscopic ex-

amination a definite delay in the passage of the barium through this segment of the esophagus was noted. Based on these findings, a diagnosis was made of an anomalous right subclavian artery originating from a left-sided aortic arch and then passing behind the esophagus in an upward oblique direction toward the right side with compression and forward displacement of the esophagus.

Figure 24 B is a reproduction of the anatomic findings as noted at autopsy, showing the retro-esophageal position of the anomalous right subclavian artery.

ANOMALIES OF THE AORTIC ARCH

A profound study of anomalies of the aortic arch and the vessels arising from it was made by Poynter in 1916.¹³ He included an excellent review with a complete bibliography covering the entire historical background up to the time of his publication.

The right aortic arch represents a persistence of the right fourth branchial artery, instead of the left fourth branchial artery, which usually forms the aortic arch in man. The right aortic arch, when present, arises from the left ventricle and courses posteriorly over the right main bronchus. It extends in front of the trachea and then passes downward as a right-sided aorta. More commonly the right aortic arch passes to the left, behind the esophagus, and then turns downward as the descending aorta, only slightly to the right of its usual position.

Among the earliest cases reported of a transposition of the aortic arch were those of Combes and Christopherson¹⁴ and of Herringham.¹⁵ In Herringham's case the aorta passed upward to the right of the trachea and crossed to the left behind the esophagus, to reach the usual position of the descending aorta on the left side of the vertebrae. The patient had complained of dysphagia and of marked dyspnea. The findings at autopsy explained the symptoms by demonstrating mechanical compression produced by the abnormally situated aorta,

which had become dilated in part of its course

In some of the cases of right aortic arch, displacement of the esophagus is produced by the pressure of a left subclavian artery originating on the right side and then passing behind the esophagus.¹¹

Apparently, the first roentgen description of a right sided position of the aortic arch is to be found in the paper by Mohr.¹ Two cases are included with the actual roentgenograms showing the aortic arch on the right side. Roentgen examination of the esophagus was not made in either case.

Illustrations of compression and displacement of the esophagus by a right aortic arch may be found in the contributions of Arkin,¹² Renander,¹ Faber, Hope and Robinson,¹³ Lowenack,¹⁴ Biedermann, Blackford, Davenport and Bayley,¹⁵ Bedford and Parkinson,¹⁶ and Gray.

The roentgen diagnosis of a right aortic arch is aided by the following features:

1. Roentgenographic examination of the chest will show an absence of the aortic arch on the left side. Instead a prominent shadow with systolic pulsations may be noted on the right side corresponding to the anatomic location of a right aortic arch.

2. There may be evidence of a pressure defect with displacement of the esophagus to the left. This pressure defect is smooth sharply defined and may cause considerable narrowing of the lumen at that level. A rounded pulsating mass may be noted to the right of the displaced and compressed segment. This evidence of pulsation caused by the right aortic arch may receive further confirmation from kymographic studies. Angiocardiographic visualization of the aorta may delineate the exact anatomic position of the arch as well as the descending aorta.

When the anomalous right aortic arch passes toward the left it will produce a smooth indentation upon the posterior wall of the esophagus with anterior displacement. However it must be borne in mind

that such a deformity may also be produced by retro esophageal pressure of a diverticulum of the left aortic arch.

When the concave pressure defect of the posterior wall of the esophagus is wide, probably it is due to a large right aortic arch or possibly a diverticulum of the left aortic arch. A shallow defect is much more apt to be produced by an anomalous subclavian artery or a ductus arteriosus. An aortic arch of smaller caliber when part of a double aortic arch may also produce a defect of lesser extent and depth than that produced by a single right aortic arch which carries the entire blood flow from the heart.

3. When visualized in the roentgenogram the trachea may show displacement to the left by the right aortic arch. The tracheogram produced by the instillation of a small amount of Lipiodol will accentuate more clearly such displacement.

4. In some cases of a right aortic arch the ligamentum arteriosum may compress the pulmonary artery against the trachea and then pass posterior to the esophagus and join the aorta. In this manner a complete vascular ring is formed which may seriously occlude the trachea and the esophagus. As a result roentgen examination of the esophagus will show not only pressure of the right lateral border but also of the left lateral border. Since the anterior portion of a double aortic arch may be much smaller than the right arch or may actually be obliterated to form a cordlike structure differential diagnosis from the constriction caused by a ligamentum arteriosum may be difficult or impossible. The subject of vascular ring formations will be discussed in greater detail later.

5. Occasionally in the case of a right aortic arch the aorta descends entirely to the right of the esophagus and does not pass retro esophageally to emerge to the left of the spine. Then the left subclavian artery originating from the right aortic arch may pass from right to left behind the esophagus. The anomalous left subclavian artery then



FIG 25 Note the anterior displacement of the esophagus by the right aortic arch. Also note the presence of diverticula.

may produce a pressure defect upon the posterior wall of the esophagus at a level slightly above the aortic arch.

A diverticulum representing a persistent remnant of the left aortic arch may be present and usually is situated behind the esophagus. The concave pressure defect on the posterior wall of the esophagus may be due to the pressure of this persistent diverticulum as well as the left subclavian artery originating from the diverticulum and passing posteriorly to reach the left side. However, the major factor in causing the concave pressure deformity will be the aortic diverticulum.

Illustrative Cases An example of a right aortic arch causing anterior displacement of the esophagus and dysphagia is illustrated in the following case.

M. H., aged 40. This patient, a woman, complained mainly of a burning sub-sternally and in her throat. There was some

difficulty in swallowing. There was no loss of weight. At times she complained of hoarseness.

Roentgen examination (Fig 25) showed marked anterior displacement of the esophagus by a right aortic arch. The course of the aortic arch in relation to the esophagus was well shown (film taken in the first, or right oblique, position). Below the anteriorly displaced portion of the esophagus was a large diverticulum springing from the anterior wall. A short distance below this area was another incompletely filled diverticulum.

The diagnosis of right-sided aorta may be aided by contrast roentgenography of the great vessels according to the technic of Robb and Steinberg.²⁶ In this manner the indirect findings of pressure on the esophagus are fortified by the more accurate visualization of the aorta itself. This is illustrated by the findings in the examination of one of our patients at Bellevue Hospital.

T. F., aged 50. The patient had entered the hospital complaining of an acute upper-respiratory infection. Examination was essentially negative, except for the teleoroentgenogram (Fig 26 A), which showed a huge sacular dilatation of either the ascending or the descending portion of the aorta, and the diagnosis of aneurysm was considered. The contrast roentgenogram (Fig 26 B) showed that the abnormal appearance was due to a right-sided aorta with elongation and tortuosity of the descending portion. This was further confirmed by roentgen examination of the esophagus (Fig 26 C) showing displacement anteriorly and to the left, produced by the anomalous position of the aorta.

DOUBLE AORTIC ARCH

A case of congenital anomaly of an aorta with a double arch has been reported by Shaw.²⁷ This is a reproduction of the arrangement usually found in reptiles. In his case the two divisions of the aorta united behind the esophagus.

The anomaly is the result of the persistence of both the right and the left fourth branchial arches. In some cases both limbs of the double aortic arch may be open, each



FIG 26 (A, *Top left*) Right sided aorta—appearance of chest (B, *Top right*) Same case Contrast roentgenogram showing the right sided aorta (C *Bottom*) Same case Displacement of the esophagus by the right sided aorta



carrying blood to the descending aorta. The two trunks are apt to be of unequal size. At times one limb of the arch usually the left is either partially or completely obliterated and cordlike in appearance.

Snelling and Erb⁴ described a case of constriction of the esophagus at the level of the third and fourth dorsal vertebrae produced by a double aortic arch. At autopsy it was found that the aorta arose in the usual manner from the left ventricle. Three centimeters from its origin the aorta divided into two. One branch passed to the left of the trachea and the esophagus and the other to the right of these structures. These two divisions united posterior to the esophagus forming the descending aorta. The esophagus and trachea were thus enclosed in a vascular ring which caused the

constriction of the esophagus seen in the roentgenographic examination

The manner in which various anomalies of the aortic arch may compress the esophagus and trachea was graphically portrayed by Gross and Ware ²⁹

Wolman ¹⁰ described a case of a double aortic arch in a newborn white male who had paroxysms of coughing and stridor. Autopsy showed a constriction of the esophagus and the trachea which had been produced by the embracement of these two structures by the double aortic arch. In addition to his own fully confirmed case his contribution includes a summary of the clinical and autopsy findings in seven other cases which during life presented a similar clinical picture of stridor, chronic cough and respiratory distress, accentuated by attempts at feeding.

In none of these cases was the diagnosis of a double aortic arch made before death.

ROENTGEN DIAGNOSIS OF VASCULAR RING FORMATIONS

In the examination to determine compression of the trachea and the esophagus by a double aortic arch the following features may be helpful. In the postero-anterior position at 90° a pressure defect may be noted on the right and the left borders of the esophagus at a level corresponding to each aortic arch. Naturally, the degree of compression will vary with the size of each arch and the degree of intimacy of the particular arch to the corresponding border of the esophagus. Examination in both the right lateral and the left lateral positions may show not only forward displacement of the esophagus but also constriction of the lumen. When more compression is exerted retro-esophageally, the constricted segment will appear much more narrowed than in the postero-anterior view. Another reason for this variation in the relative degree of constriction is the fact that the pressure from behind the esophagus is apt to flatten the posterior wall against the anterior wall, thus causing a greater

spread in the lateral dimension of the involved segment than in the postero-anterior direction.

Sometimes the associated compression of the trachea may be seen in films taken with sufficient technical skill to show the outline of the soft tissues of this region ²⁵. Considerable help in the visualization of the constricted trachea may be obtained by the direct instillation of a small amount of Lipiodol or a Lipiodol spray into the trachea ¹¹.

A vascular ring produced by the junction of a right aortic arch and a left ligamentum arteriosum may also constrict the trachea and the esophagus abnormally. In this group of cases the aortic arch is on the right side and is continuous with a right descending aorta. The ligamentum arteriosum originates from the pulmonary artery to the left of the trachea and the esophagus. It then passes behind the esophagus, finally joining the thoracic aorta on the right side just below the level of the aortic arch ³¹.

The following roentgenographic features may aid in establishing the diagnosis. The aorta may be noted on the right side. The barium-filled esophagus in the postero-anterior position may show a pressure defect on the right side. On the posterior wall of the esophagus a slight indentation may be present, caused by the constriction of the ligamentum arteriosum as it passes behind the esophagus from its point of origin at the pulmonary artery on the left side to the aortic arch on the right side.

The pressure defect produced by the ligamentum arteriosum is at a lower level of the esophagus than the pressure deformity produced by the right aortic arch against the right lateral border of the esophagus in the postero-anterior position. In the event of a wide defect on the posterior wall this may be the result of the constriction produced not only by the ligamentum arteriosum but also, in part at least, by the retro-esophageal position of the right aortic arch. Further help may be obtained by the demonstration of an indentation on the right

side of the trachea when outlined by the direct introduction of a small amount of Lipiodol

This evidence of a pressure defect upon the right side of the visualized trachea is only an additional aid in the demonstration of a right aortic arch but does not necessarily indicate vascular ring formation by means of the ligamentum arteriosum

A difficulty in differential diagnosis arises in the presence of the rare type of anomaly of the aorta described by Paul.³ This anomaly consists of a left aortic arch which then passes behind the esophagus to become continuous with a right sided descending aorta. Therefore radiographic examination of the barium filled esophagus in the postero-anterior position then shows an indentation on its left border similar to that noted in the case of a left aortic arch. The right and the left anterior views show the concave indentation upon the posterior wall produced by the retro esophageal position of the aortic arch. The descending aorta then continues downward to the right of the esophagus.

In the vascular ring described by Edwards³³ the aortic arch originating on the left side passed to the right behind the esophagus and then continued in a downward direction on the right side. The ligamentum arteriosum arose on the right side from the lower anterior aspect of the aortic diverticulum which lay against the right posterior aspect of the esophagus. It then extended to the upper and proximal aspects of the right pulmonary artery thus forcing the proximal portion of the right pulmonary artery and the bifurcation of the pulmonary trunk against the anterior portion of the trachea. Thus a vascular ring was formed which encircled the trachea and the esophagus. The ring consisted of the aortic arch on the left side, the retro esophageal portion of the aorta, the ligamentum arteriosum on the right side and anteriorly, the right pulmonary artery and the bifurcation of the pulmonary trunk.

These anatomic findings were found at

autopsy in the case of a 17 month old boy who died of small bowel obstruction, which was the result of adhesions secondary to an operation when three days old for an imperforate anus. Dysphagia had been noticed when the child began eating solid food.

In one of the cases reported by Wurtz and Powell³⁴ a newborn, full term, white male infant exhibited cyanosis, respiratory embarrassment and considerable secretion of mucus from the nasal pharynx. Autopsy revealed a right aortic arch which extended behind the esophagus and the trachea. It then emerged to the left and posterolateral to these structures where it was joined by the ductus arteriosus. Thus a vascular ring was formed which constricted the trachea and the esophagus and apparently was the cause of the clinical symptoms. The descending aorta was on the left side.

Carson and Goodfriend³ showed an unequal but bilateral indentation of the barium filled esophagus in the postero-anterior view caused by the constricting vascular ring of a double aortic arch confirmed at operation. An anterior and a posterior constriction of the esophagus were also demonstrable in the right anterior oblique position. The tracheogram obtained by the introduction of Lipiodol also showed a bilateral constriction just above the bifurcation.

A vascular ring causing obstruction of the esophagus and the trachea is illustrated by the case of the newborn female infant Baby De C. Cyanosis was noted as soon as feeding by mouth was begun. In between feedings the child showed respiratory difficulty and she was transferred to the pediatric service. Examination showed that the infant was in marked respiratory distress accompanied by stridor. The patient seemed to be more comfortable when lying with the head in hyperextension. Stridor increased when the head was flexed. During feeding the respiratory embarrassment increased and the child appeared to be cyanotic. Physical examination was otherwise negative except for a few rales in the chest.

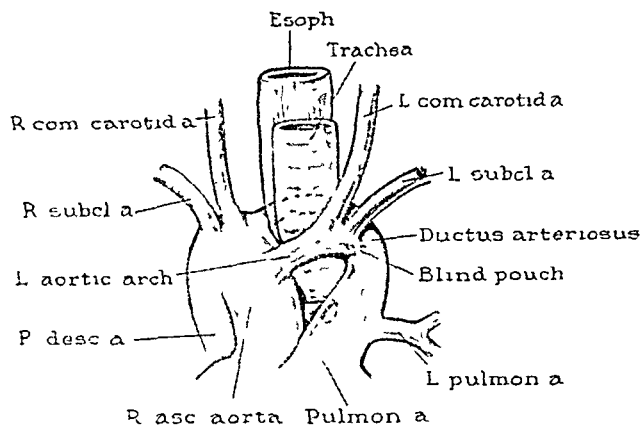


FIG 27 (A, *Top, left*) The vascular ring which occluded the trachea and the esophagus, reconstructed on the basis of the autopsy findings (B, *Top, right*) Postero-anterior position at 90° This shows the constriction of the esophagus at the level of the vascular ring Note that the larger defect is on the left side, although the aortic arch was found to be on the right side That part of the ring on the left side apparently was in closer apposition to the esophagus and therefore produced a more marked pressure deformity than the right aortic arch which presumably during life had compressed the right lateral border of the esophagus to a lesser degree (C, *Bottom left*) Examination in the right oblique position shows the extreme degree of constriction of that segment of the esophagus incorporated within the vascular ring (D, *Bottom, right*) A similar degree of constriction of the esophagus is also noted in the left oblique position Therefore, the compression of the esophagus was much more marked postero-anteriorly than laterally

Based on the clinical picture as well as the radiographic findings which will be discussed later, a diagnosis was made of a vascular ring constricting the esophagus and the trachea. The presence of the vascular ring was confirmed at operation, and the ring was severed.

Autopsy revealed the exact anatomic components of this vascular ring (Fig 27 A). The heart itself was entirely normal, as was the pulmonary artery. The ascending aorta and the descending aorta were on the right side. The first vessel that branched off crossed the midline and gave origin to the left carotid and the left subclavian arteries. This vessel, the left (anterior) aortic arch, seemed to communicate with a large vessel (the patent ductus arteriosus). However, on opening both structures there appeared to be no real communication. The large ductus arteriosus was almost a continuation of the pulmonary artery and passed behind the trachea and the esophagus to join the right aortic arch, thus completing the aortic ring. Therefore, the vascular ring was formed by the following structures:

- 1 On the right side, by the right aortic arch
- 2 Posteriorly by the ductus arteriosus
- 3 On the left side, by the ductus arteriosus and part of the left aortic arch
- 4 Anteriorly by the left aortic arch

The radiographic examination showed the constriction produced by the vascular ring. Figure 27 B shows the appearance of the esophagus in the postero-anterior position. There is some constriction of the esophagus at about the level of the aortic arch with a pressure defect on either side. However, the major defect is on the left side. Because of this finding it was believed that the aortic arch was on the left side and not as the case proved to be at autopsy a vascular ring with the aortic arch on the right side. Perhaps this may be explained by assuming that the vascular structures on the left side were able to exert more of a pressure defect. The right aortic arch itself may not have

been in very close proximity to the right border of the esophagus and therefore produced less evidence of compression. Figure 27 C (in the right oblique position) shows the extreme degree of constriction of that portion of the esophagus trapped by the constricting vascular ring. Figure 27 D (left oblique position) also shows evidence of the marked constriction of the esophagus produced by the vascular ring. A study of the appearance of the esophagus in these different views shows that the major degree of occlusion due to the constriction of the vascular ring was in the postero-anterior direction. There appeared to be much less constriction of the esophagus laterally.

ANOMALIES OF FORMATION

Types of Congenital Malformation
Ballantyne³⁶ in his classic work has divided congenital malformations of the esophagus as follows:

- 1 Complete absence of the esophagus, its place being taken by a thin muscular band stretching from the pharynx to the cardiac end of the stomach
- 2 Termination of the esophagus in a simple cul de sac
- 3 Termination of the esophagus in a cul de sac with the trachea or bronchi
- 4 Tracheo-esophageal fistula, the esophagus being otherwise normal
- 5 Membranous obstruction of the esophagus
- 6 Presence of esophageal diverticula
- 7 Duplicity of the esophagus

When the embryo is about 4 mm long there are two lateral indentations, one on each side of the entodermal pouch. These gradually become deeper, finally meeting in the center, thereby forming a septum which divides the pouch into an anterior and a posterior tube. This septum formation is complete when the embryo is 11 mm in length. The respiratory system develops from the anterior tube and the alimentary tract from the posterior tube. It is believed that as a result of the incomplete development of the septum, a fistulous communi-

cation may remain between the trachea and the esophagus

During this period some important changes are taking place in the esophagus. At first the lumen is obliterated by a proliferation of the epithelial lining, thereby resulting in the formation of a solid, cord-like structure. Later vacuoles develop within this solid structure. These vacuoles gradually coalesce, re-establishing the lumen. Should there be an arrest of the process of vacuolization and coalescence of these vacuoles a complete stenosis or atresia of the affected segment will result. Also, should the ingrowth of mesoderm fail to effect complete separation of the esophagus from the trachea then the atresia will be associated with a tracheo-esophageal fistula.

COMPLETE ABSENCE OF THE ESOPHAGUS
One of the earliest cases of complete absence of the esophagus is to be found in the book by Billard³⁷ published in 1828.

Aug. 8, 1820, Madam — gave birth to a child at term that appeared to be normal. The child was given a little sugar water which he swallowed greedily but which regurgitated immediately through the mouth and nose almost suffocating the child. This recurred with each attempt at feeding and from this it was easy to surmise the presence of a malformation of the esophagus. The child lived for a week and died of starvation.

At autopsy

the stomach was adherent to the diaphragm.

The esophagus was entirely missing and the pharynx ended in a cul-de-sac.

DOUBLE ESOPHAGUS Blasius,³⁸ in 1677, in *Observationes Medicae Rarioris*, described a case of duplication of the esophagus under the caption of a divided esophagus.

In an infant of 5 years on the tenth of April, 1670, we found an esophagus for the greatest part divided from the level of the first to the sixth rib. Thus is given here a double pathway for the swallowed material. Yet the duct here was single in the beginning and then again when it joined the stomach. This was similar to the case of a recently born infant that I had previously described

(The findings are illustrated by a drawing, Fig. II, Plate VI, pg. 113.)

Habershon³⁹ described the case of a Cyclopean monster in which the viscera of a double fetus existed in a single peritoneal cavity. There was a double esophagus united in a single stomach.

A remarkable case of double esophagus is to be found in the excellently documented report by Gjorup.⁴⁰ The story was of a little girl 17 days old who had lost weight persistently since birth. There was no vomiting. Physical examination revealed considerable abdominal protrusion due to a palpable mass. On exploration the stomach was found to be greatly dilated and surrounded by adhesions. An attempt was made to sever these adhesions, but the operation had to be curtailed because of respiratory embarrassment.

Autopsy showed two distinct esophaguses, both entering the abdominal cavity through the same opening in the diaphragm but entering the stomach separately. Water was injected separately into each esophagus. An additional remarkable fact was then discovered. The water introduced into the right esophagus filled out a part of the stomach, which was able to empty itself by way of the pylorus. However, there was no communication between this part of the stomach and the left cavity of the stomach. Water introduced by way of the left esophagus entered this left cavity of the stomach, which, however, was unable to empty itself of its fluid, either by way of the pylorus or by way of any communication with the right cavity of the stomach. Therefore, Gjorup's case was not only one of a double esophagus but also of a double stomach. Excellent photographs showing these strange findings at autopsy accompany the description.

A case of a double esophagus, with carcinoma involving one, was reported by Butler and Ende.⁴¹ The patient was a male, aged 49, with a mass on the left side of the neck which on biopsy proved to be an undifferentiated metastatic carcinoma. He also com-

plained of dysphagia. At autopsy the mediastinal structures were removed en masse. Microscopic sections revealed two separate esophageal lumens. The more anterior one was lined by normal epithelium except for small areas invaded by tumor. The posterior lumen was lined almost entirely by cancer cells. Following this observation the gross specimen was re-examined, and the presence of two separate esophageal lumens was demonstrated, each opening into the stomach. The anteriorly placed esophagus joined the stomach in the usual manner. The posteriorly placed esophagus, the wall of which was invaded by carcinoma, extended along the greater curvature of the stomach and opened into it 6 cm distal to the opening of the anterior esophagus.

MEMBRANOUS OBSTRUCTION OF THE ESOPHAGUS. A rare congenital anomaly capable of causing stenosis of the esophagus is a membranous diaphragm which may have an opening within it. This anomaly may be explained on the basis of insufficient vacuolization of the esophagus with incomplete restitution of the lumen during fetal development.

Morrell Mackenzie⁴ referred to several of the early cases in the literature of membranous obstruction of the esophagus (Rossi 1826).⁴³ He described the autopsy findings of another case that had been reported in 1851.⁴⁴

About 6 fingers' breadth below the pharynx there was a completely circular valve, with an opening about one cm in diameter. This valve seemed formed by a folding inwards transversely of the mucous membrane, involving the whole circumference of the tube the free edge of the valve being strengthened by firm tendinous fibres running around it.

Clark⁴ reported a case of a congenital web of the esophagus in 1911. The story was that of a female aged 24 who since early childhood had to chew her food into very small pieces in order to avoid difficulty in swallowing. She had repeated attacks of dysphagia of increasing severity brought on by attempts at swallowing any solid food.

Finally, difficulty was encountered even with liquids. Endoscopy revealed a membrane at about the region of the mouth of the esophagus. A little to the left of center of this membrane was a small opening 3.5 mm in diameter which communicated with the esophagus. This 'hymen' was ruptured by the introduction of the esophagoscope with clinical cure of the dysphagia. In discussing Clark's case Harris P. Mosher stated that a few months previously he had encountered a case of a true web of the esophagus in a patient with a 15 year history of dysphagia. Endoscopic examination revealed a diaphragm with a small central opening about 1 inch distal to the cricoid cartilage. The patient was cured by instrumental dilatation.

In 1928 Beatty⁴⁶ made an excellent survey from the literature of the findings in 50 cases of congenital stenosis of the esophagus. In addition he included 2 cases from his own personal experience, 2 other cases in the literature not previously reported, as well as the appearance of the stenosis in 3 museum specimens. In quite a number of these cases a congenital membrane was the cause of the esophageal stenosis. The membrane might be partial or complete and was present at the beginning of the esophagus immediately posterior to the cricoid cartilage or at its distal end a short distance above the cardia.

Abel⁴⁷ noted that the diaphragm may be partial or complete and may be the only abnormality of the esophagus causing obstruction. In his own case of dysphagia in a newborn child, esophagoscopy disclosed a glistening membrane completely occluding the esophagus about 1 inch below the level of the bifurcation of the trachea. The membrane was ruptured by the pressure of the esophagoscope and normal esophageal mucosa was seen distal to it. The infant made a good clinical recovery, although later roentgenographic studies showed some persistent narrowing of the distal end of the esophagus. Also, it is interesting to note that this infant developed a hypertrophic pyloric

stenosis 4 weeks later, for which she was operated on successfully

Guthrie⁴⁸ described the autopsy findings in a case of congenital esophageal stenosis in an infant. There was a small semicircular diaphragm at the pharyngo-esophageal junction, in addition to a stricture 1 inch in length, high up in the esophagus.

Atresia The most serious congenital anomaly of the esophagus is that produced by atresia. This condition may be associated with a fistulous communication with the trachea. The comparative rarity of the disease is shown by the fact that only 9 cases of atresia of the esophagus were found in our Bellevue Hospital series of 22,810 autopsies from 1905 through 1935.

Atresia of the esophagus with tracheo-esophageal fistula in the embryo has been described by Plass,⁴⁹ Ysander⁵⁰ and Gruenwald.⁵¹ Ysander's case occurred in an embryo monster 8 mm in length, of which he showed a model demonstrating a double esophagus and trachea on each side, with a bilateral reduplication of atresia with tracheo-esophageal fistula. The upper part of the esophagus on each side ended in a blind pouch. The lower portion of the esophagus communicated with the corresponding trachea. Both esophagi then entered a common stomach.

ATRESIA WITH TRACHEO-ESOPHAGEAL FISTULA An early description of esophageal atresia with tracheo-esophageal fistula is to be found in the work by Thomas Gibson in 1703 in his book having the following quaint title: *The Anatomy of Humane Bodies Epitomized wherein All the Parts of Man's Body, with their Actions and Uses, are Succinctly Described According to the Newest Doctrine of the most Accurate and Learned Modern Anatomists*.⁵²

The now generally received opinion of the Foetus's receiving nourishment by the mouth in the latter months, may besides the reasons above recited from Diemerbroeck, be further confirmed by the following Observation. About November, 1696, I was sent for to an Infant that could not swallow. The Child

seemed very desirous of food, and took what was offered it in a Spoon with greediness, but when it went to swallow it, it was like to be choaked, and what should have gone down returned by the mouth and nose, and it fell into a struggling convulsive sort of fit upon it. It was very fleshy and large, and was two days old when I came to it, but the next day died. The Parents being willing to have it opened I took two Physicians and a Surgeon with me. Opening the Abdomen first, the Guts had some of the Meconius remaining still in them, though the child had gone two or three times to stool. The stomach had in it a pretty deal of a slimy sort of Liquor, (or jelly rather) somewhat like pretty thick (strained) water-gruel. I shall not mention any observations upon other parts in the Abdomen, as being not to our present purpose.

When we cut open the Thorax, and taking out the Gullet (with the Wind-pipe, Lungs, etc.) continued to the Stomach we blew by a pipe down the Gullet, but found no passage for the wind into the Stomach. Then we made a slit in the Stomach, and put a pipe into its upper orifice, and blowing we found the wind had a vent, but not by the top of the Gullet. Then we carefully slit open the back-side of the Gullet from the stomach upwards, and when we were gone a little above half way towards the pharynx, we found it hollow no further. Then we began to slit it open from the pharynx downwards, and it was hollow till within an inch of the other slit, and in the imperforated part it was narrower than in the hollowed. This isthmus (as it were) did not seem ever to have been hollow, for in the bottom of the upper, and the top of the lower cavity there was not the least print of any such thing but the parts were here as smooth as the bottom of an acorn cup.

Then searching what way the wind had passed when we blew from the stomach upwards, we found an oval hole (half an inch long) on the fore-side of the gullet opening into the aspera arteria a little above its first division, just under the lower part of the isthmus of the above mentioned.

Now, I say this is a plain confirmation of the foetus's being nourished by the mouth, for the gula being impervious, nature had formed this hole in the wind-pipe and gullet, for the liquor contained in the amnios to pass into the Stomach, which it might do without prejudice, or any fear of choking, in the Womb, while the Child breathed not but

when it was born and came to breathe there could be no longer any passage this way and so the Infant was necessarily furnished

Martin's case of tracheo-oesophageal fistula with atresia is described in the proceedings of the Societe Royale de med de Marseille in 1820 is extremely interesting³

A newborn child rejected all liquids and died in 36 hours Martin did an autopsy The stomach was exposed An attempt was made to fill it with air by means of a sound introduced into the pharynx The efforts were useless The sound was then placed in the trachea Air was blown into it, but to the great astonishment of M Martin the air entered the stomach and it became distended at the same time as the lungs Then a careful dissection was made The oesophagus was vainly sought for but it did not exist being replaced by a cul de sac a few inches long A probe was then introduced into the trachea and here the astonishment was redoubled At the level of and in the space between the two bronchi this probe penetrated into an elastic membranous tube of the caliber of a little quill and through it the stomach could be reached The two bronchi were normal throughout their entire extent The repeated vomiting and the prompt expulsion of liquid were explained by this singular arrangement

Another of the earliest recorded cases of atresia of the oesophagus with tracheo-oesophageal fistula is to be found in the report by John Houston under the heading of "Pathological Observations appearing in The Dublin Hospital Reports and Communications in Medicine and Surgery in 1830"

In the month of November 1828 Mrs T was delivered of a male infant The labour was natural and the child healthy and of the full size it exhibited no outward imperfections except a misplacement of the orifice of the urethra which was situated underneath and behind the glans penis (A similar malformation of the urethra had been noticed in an elder boy of the same family)

On every attempt to suck fits of coughing immediately supervened threatening suffocation by their violence and lasting until the milk was all disgorged again Similar consequences followed every attempt at feeding the infant from a spoon When only half a tea

spoonful was swallowed no signs of inconvenience were manifested, but as soon as the remaining half was taken, all the symptoms above enumerated supervened

On the suspicion that a stricture of the oesophagus or pharynx might exist, an attempt was made to pass a bougie by the mouth into the stomach, but without effect the instrument met with obstruction at a short distance from the mouth, and could not by any means be guided onwards All further attempts therefore, either at the use of instruments or the administration of food by the mouth, were desisted from, as they only caused distress, or brought on suffocation which threatened the patient's life The infant quickly became emaciated it cried incessantly, and died on the following day

At the invitation of Dr C Johnson I assisted in making the dissection The mouth, tongue and opening of the fauces were naturally formed The pharynx was unusually wide and terminated some way down the neck in a cul de sac without having any connexion with the oesophagus The larynx and its muscles were all perfect The trachea was also complete on the anterior and lateral parts, but the posterior wall was perforated by a wide opening, from which the oesophagus took origin, and which appeared so smooth and oblique as to render the passage from the larynx into that tube as direct and easy as that along the trachea into the lungs

Excellent reproductions of the pathology accompany the description The condition was also described at about the same time (1829) by Andral⁵

An excellent illustration of a tracheo-oesophageal fistula of the lower segment is to be found in the remarkable atlas by Vrolik, published in 1849

Figures 2 and 3 show a congenital defect observed by the very famous Tilanus in the cadaver of an infant recently born who lived as long as five days after birth The infant although it lived for this length of time could not swallow because the oesophagus was terminated a short distance below the isthmus of the fauces in a blind sac Anatomical examination showed the pharynx and larynx to be well formed but the very small oesophagus above the level of the bronchi terminated in a blind sac and then began again and continued on its way to enter the stomach On the posterior wall of the trachea there was an oblong,



FIG. 28, Atresia of the esophagus.



FIG. 29, Atresia of the esophagus. Note the gas in the alimentary tract.

rounded foramen which communicated with the esophagus. It appears therefore that there are two congenital abnormalities. . . . The esophagus shows an intrinsic atresia. . . . In addition the esophagus and trachea form a single canal.

Kelth⁶⁷ described cases of marked narrowing of the beginning of the esophagus shallow to a fibrous stenosis of the pylorus. The circular muscle was atrophied and was contracted and replaced by loose connective tissue. There was no clear evidence of dentilization. He also described cases of congenital stenosis of the esophagus in which the part that was in the posterior mediastinum was reduced to a fibrous, imperforate cord.

Atresia of the esophagus may be present

without any associated tracheal fistula. When a fistula is present, the communication may be between the upper segment of the esophagus and the trachea or between the lower segment and the trachea, or the trachea may communicate with both upper and lower segments. The commonest variety is the one which exhibits a fistula between the terminal portion of the esophagus and the trachea.

Röntgen Diagnosis of Atresia of the Esophagus. When atresia of the esophagus is suspected, examination by the introduction of a barium suspension is contraindicated since it may spill over from the blind pouch into the trachea and the lungs. The

corroboration of the clinical impression of atresia may be accomplished by the introduction of a small nasal catheter and its arrest noted by roentzenographic examination. Air may be introduced by means of a syringe and the distended sharply delimited portion of the esophagus may be visualized in this manner. A small amount of Lipiodol may be introduced through the nasal catheter under fluoroscopic control and when the roentgen examination has been completed it may be promptly aspirated.

When an examination is made the appearance of the abdominal cavity should also be observed and included in the roentgen examination of the chest. When a fistula exists between the trachea and the distal end of the esophagus large amounts of air will be found distending the stomach and the intestine having reached the alimentary tract by way of the inspired air. If the fistulous communication is limited to the upper blind pouch there will be no air in the alimentary tract. Such absence of air however does not necessarily always preclude the existence of a fistula between the trachea and the distal esophagus. An unusually small communication between the trachea and a narrowed segment of the esophagus may prevent the entrance of air.

Illustrative Cases. Congenital atresia of the esophagus is illustrated by the following case.

B. P. (Fig. 28). This child had been born 12 days previously. Since birth it had not been able to retain its feeding, the food being immediately regurgitated. A gastrostomy was done. About $\frac{1}{2}$ ounce of sterile water was given through the gastrostomy tube. The child became cyanotic, started to gag and was in poor condition for the next 5 minutes. This was very suggestive of a communication of the lower end of the esophagus with the trachea. A second attempt about an hour later produced even more severe attacks of cyanosis with cessation of respiration which was as physical in character.

The autopsy report stated that the esophagus is divided into two portions which are entirely separate and distinct. The first is an

upper portion which is a tube with a sacular end. This lower end is entirely closed and terminates at the level of the bifurcation of the trachea. From the dilated closed end there comes forth a slender strand of fibrous tissue. This separate and distinct upper portion of the esophagus does not communicate in any way with the trachea and its mucosa appears entirely normal. The lower portion of the esophagus comprises the inferior two thirds; it begins at the bifurcation of the trachea and terminates at the cardiac opening of the stomach. The origin comprises the tracheo-esophageal fistula and except for a very slight curvature where the esophagus blends with the bifurcation of the trachea the two structures might well be considered as a continuous straight tube. Thus when a probe is passed through the gastrostomy it passes through the cardiac opening of the stomach up the inferior portion of the esophagus out through the trachea and emerges from the larynx.

From the above description it may be seen readily that there is no possibility that the food taken by mouth will enter the stomach and there is a great possibility that food given by way of the gastrostomy will enter the bronchi and the lungs. In fact the lungs present a rather speckled grayish white appearance, the white areas being a few millimeters in size and surrounded by congested lung tissue. These white areas appear to represent areas of asphyxiated barium. A careful search of the other viscera failed to reveal any other congenital anomalies.

Roentgen examination of the esophagus following the introduction of Lipiodol by way of the mouth showed complete obstruction of the esophagus at the level of the third rib. There was moderate dilatation of the esophagus above this point. Under fluoroscopic examination Lipiodol was injected into the stomach through the gastrostomy tube. The stomach was very small and the Lipiodol passed over into the coils of small intestine. A barium mixture was then introduced into the stomach. The barium passed into the esophagus to the level of the fourth dorsal vertebra, which portion was about 1 inch below the lowermost portion of the closed-off esophagus as shown by the residual Lipiodol injected the preceding day. At this point the barium passed into the bronchial tree and soon spread into the minute ramifications of the lungs. (Fig. 28).

The diagnosis was as follows: (1) atresia of the esophagus at the level of the third dorsal vertebra. (2) esophageal tracheal fistula, the fistula occurring between the lower portion of the esophagus and the trachea.

The following is an example of atresia of the esophagus with air in the alimentary tract

A K, infant, 11 days The infant was brought to the hospital because it had been regurgitating its food since birth

Operation revealed an atresia of the proximal esophagus and a tracheo-esophageal fistula involving the distal segment of the esophagus The gap between the two ends of the esophagus was too great to permit anastomosis Therefore, the proximal end was brought out to the surface of the skin The distal end of the esophagus was tied off A rubber tube was inserted into the stomach A gastrostomy was done

A small amount of Iodochloral (27 per cent iodine in refined peanut oil) was instilled into the esophagus through a rubber catheter, and a roentgen examination was made Figure 29 shows the blind termination of the upper segment of the esophagus A small amount of the Iodochloral entered the trachea Gas is present throughout the alimentary tract

The diagnosis was atresia of the esophagus

with an upper blind pouch The presence of gas in the intestine indicated that there was a communication between the distal segment of the esophagus and the trachea (tracheo-esophageal fistula) These findings were confirmed at operation, as noted above.

In the next case of atresia of the esophagus the fistula communicated with the right main bronchus This premature female infant (E S), one of twins, regurgitated everything from birth, and nurses noted that the gavage tube was passed with difficulty, and that the feeding returned immediately by way of the mouth and the nose

Operation revealed that the proximal end of the esophagus consisted of a blind pouch which terminated at about the level of the third dorsal vertebra There was a fistulous communication between the distal segment of the esophagus and the right main bronchus

Roentgen examination (Fig 30) shows the arrest of the Lipiodol in the blind pouch



FIG 30 Atresia of the esophagus

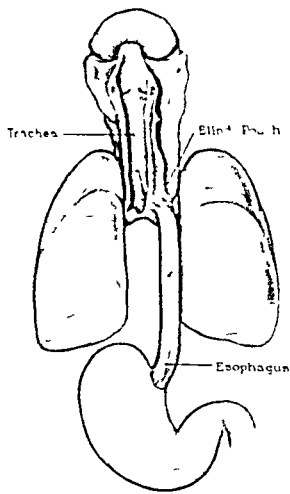


FIG 31 (A *Left*) Atresia of the esophagus. The proximal segment is distended with Lipiodol. A small amount has escaped into the trachea and the bronchi. Gas is present in the small intestine. (B *Right top*) Anatomic appearance at autopsy showing the blind proximal segment of the esophagus and a probe in the distal segment of the esophagus passing through the communicating fistula into the trachea. (C *Right bottom*) Semidiagrammatic reproduction of the appearance at autopsy viewed posteriorly.

formed by the proximal portion of the esophagus. A small amount has spilled over into the bronchial tree. Gas was noted in the stomach and the intestine. The preoperative diagnosis was atresia of the esophagus with a fistula between its distal portion and the trachea. The diagnosis was confirmed at operation as already noted except that the fistula communicated with the right bronchus.

The following is a completely documented case of atresia of the esophagus with tracheo-esophageal fistula including a photograph of the autopsy specimen.

B. L. newborn. This spontaneously delivered well-developed infant at birth showed

evidence of the escape of a large amount of secretion from the nose and the mouth, as well as cyanosis. The infant also regurgitated the administered food. A small catheter passed into the esophagus met with obstruction at a distance of 4 inches, indicating an atresia. The exact anatomic location of the atresia was shown by roentgen visualization after filling the proximal segment of the esophagus with Lipiodol (Fig 31 A). A small amount escaped into the tracheobronchial tree. Gas was noted in the intestine, thus indicating a fistulous communication between the lower segment of the esophagus and the trachea. The appearance of the specimen at autopsy may be noted in Figure 31 B. The upper segment of the esophagus is well shown. A probe is present extending through the communication between the distal segment of the esophagus and the trachea. Figure 31 C is a semidiagrammatic reproduction of the findings at autopsy viewed posteriorly.

A successful repair of an atresia of the esophagus is illustrated in the following case.

A P. This was a 1-day-old female infant, born at another hospital full term, following a normal uneventful delivery. The infant was transferred to Bellevue Hospital because of

copious frothy mucus discharge from the mouth and the immediate regurgitation of dextrose-water feeding. The infant seemed to be cyanotic during the feeding.

Physical examination was essentially negative. On admission a rubber catheter, passed through the nose under fluoroscopic control, encountered an obstruction in the esophagus so that it could not be passed into the stomach. Lipiodol was injected through the catheter, and a blind pouch was noted in the upper portion of the esophagus.

Radiographic examination (Fig 32 A) showed the upper pouch of the esophagus. A considerable amount of air was present in the stomach and the intestines. The preoperative diagnosis was atresia of the esophagus and a fistula between the distal end of the esophagus and the trachea or bronchus. Operation confirmed the diagnosis of atresia. A fistula between the trachea and the distal segment of the esophagus was found. This was dismantled. A primary anastomosis was performed between the upper and the lower segments of the esophagus. The postoperative course was remarkably uneventful. The patient gained weight and was placed on regular feedings. One month later radiographic examination (Fig 32 B) disclosed continuity of the lumen of the esophagus. There was a moderate degree of constriction at the site



FIG 32. Atresia of the esophagus with tracheo-esophageal fistula and successful operative repair. (A, Left) Appearance prior to operation, showing the blind upper pouch as well as the gas in the stomach and the small intestine. (B, Right) Appearance after operation, showing continuity of the lumen of the esophagus.

of anastomosis. This, however, was insufficient to interrupt the flow of barium through it.

Tracheo-esophageal Fistula Without Atresia A rare congenital anomaly is tracheo-esophageal fistula without atresia. The earliest description which I have been able to find in the literature of a tracheo-esophageal fistula without atresia is that by Lamb in 1873.¹ The story was that of a newborn infant who died at the age of 7 weeks. Attempts at nursing had caused suffocation. At autopsy

in the median line, nearly half an inch below the lower border of the cricoid cartilage, was a fistulous communication between the two tubes having a longitudinal diameter of three lines and a transverse diameter of one line. The direction of the fistula was downward and backward, the opening in the esophagus being at a lower level than that in the trachea; the edges were smooth and rounded and the mucous membrane was normal. The danger of passage of contents of the esophagus into the trachea appears to have been guarded against to some extent by the close apposition of the walls of the fistula.

This specimen was contributed to the Army Medical Museum and is exhibited as Number 1161 of the Medical Section. To show the fistula more distinctly a whalebone probe was introduced and the hardening action of the alcohol used as a preservative fluid has rendered the fistulous opening permanently dilated.

There was no evidence of an associated atresia, and he definitely differentiated this type of congenital fistula from those cases of imperforate esophagus with or without fistulous communication with the air passages.

In the same year (1873) Pinard⁶⁰ described a similar case of tracheo-esophageal fistula without atresia. The newborn took the breast very well but after a few attempts at sucking had an attack of suffocation and vomited the ingested milk. The face became livid and breathing was painful and embarrassed. Pure meconium issued spontaneously from the urinary opening.

In addition to an atresia of the rectum with recto-urethral fistula the child was

considered to have a probable perforation of the esophagus. When a sound was passed into the pharynx and the esophagus it met without obstruction. Autopsy, in addition to confirming the congenital anomaly of the rectum, showed a fistulous communication between the trachea and the esophagus, measuring about 3 cm in length. The esophageal mucosa of the fistula was continuous with the tracheal mucosa. The rest of the digestive tract was normal.

In Imperatori's case⁶¹ of tracheo-esophageal fistula without atresia the intercommunicating fistula was slitlike in appearance. Its existence was demonstrated by esophagoscopy and later confirmed by x-ray studies. Imperatori was able to pass a urethral catheter through the tracheal fistula into the stomach, following which a roentgenogram was taken. The fistula was closed off successfully by surgical intervention.

Gegenbrach and Dobos⁶² included a case of tracheo-esophageal fistula without atresia confirmed by autopsy. Leven and Lannin⁶³ found two such cases in the records of the University Hospital and the department of pathology of the University of Minnesota in 22 years, from 1916-1938 inclusive.

Haight⁶⁴ reported two cases of congenital tracheo-esophageal fistula without atresia in a series of 65 cases of anomalies of the esophagus at the University of Michigan Hospital. In one case the child was 4 years old at the time of operation for this condition and it is rather remarkable that it was able to maintain a fair degree of health in spite of so serious a lesion. The preoperative diagnosis was established by the fact that a catheter could be passed readily through the entire length of the esophagus into the stomach. However, when iodized oil was swallowed, it entered the bronchial tree from the esophagus thus establishing the diagnosis of a fistulous communication. The diagnosis was further confirmed by tracheoscopy.

Haight called attention to the importance of examination of the patient in the prone



FIG 33 Tracheo-esophageal fistula without atresia (A, *Top, left*) Appearance of chest prior to examination of the esophagus (B, *Top, center*) Visualization of the esophagus after the introduction of the Lipiodol, showing absence of atresia (C, *Top, right*) Visualization of the tracheobronchial tree after the introduction of 2 cc of Diodrast into the upper portion of the esophagus, demonstrating a tracheo-esophageal fistula. It is of technical significance that the fistula was not demonstrated successfully following the injection of Lipiodol. It is assumed that this may have been due to the watery character of the Diodrast which enabled it to permeate through the fistulous opening more readily than the viscid oily Lipiodol (D, *Bottom, left*) Autopsy specimen, showing the opening of the fistula on the esophageal side. There was no other evidence of pathology of the esophagus (E, *Bottom, right*) Opening of the fistula as viewed from the trachea.

position after the administration of Lipiodol in order to aid its escape through the fistula into the trachea which otherwise might not take place.

Swenson in 1948,⁶⁵ included radiographic

evidence of a case of tracheo-esophageal fistula without atresia.

Abbott and Hopkins⁶⁶ had three cases of tracheo-esophageal fistula without atresia. One of the cases was well documented by

means of a roentgenogram in the left lateral position showing the entire esophagus well outlined by means of Lipiodol as well as the fistulous tract between it and the trachea.

ROENTGENOGRAPHIC EXAMINATION. In such cases of tracheo-esophageal fistula without atresia of course the catheter introduced into the esophagus will pass readily into the stomach. When a fistula between the esophagus and the trachea is suspected the catheter may be withdrawn to about the level of the first dorsal vertebra and a small quantity of Lipiodol (0.5 to 1 cc) is introduced. With the child's face down perhaps aided by a slight Trendelenburg position, the Lipiodol injected through the catheter may find its way more readily from the esophagus through the fistula into the trachea. This would be particularly true if as in the experience of Abbott and Hopkins the fistula was at a higher level in the trachea than in the esophagus. This anatomic peculiarity may also explain why in some cases the newborn with this type of lesion may survive without prompt suffocation. Since the fistulous tract runs in an upward and cranial direction from the esophagus to the trachea much of the swallowed food may enter the stomach without escaping into the lungs. Examination in the lateral position may be extremely useful in showing the fistula bridging the area between the trachea and the esophagus. It is possible that Lipiodol may fail to enter a very small fistulous opening in which event a watery solution such as Diodrast may be more successful as will be noted in the case about to be described.

The following is an account of my personal experience with a case of tracheo-esophageal fistula without atresia encountered at Bellevue Hospital. This case was previously reported by Cardulo and Berens.⁶⁷

R. B., male, was transferred to our pediatric service 48 hours after birth with a history of vomiting all ingested fluids and of an attack of cyanosis. The clinical impression was

pneumonia and possible esophageal atresia. Physical examination corroborated by radiographic study of the chest showed evidence of a diffuse bronchopneumonic process. The heart, the trachea and the mediastinal contents were markedly retracted into the left hemithorax. These findings were believed to be due to inspiration pneumonia secondary to esophageal atresia. A study of the skeletal structure at the same time disclosed an anomalous development of the fourth and the fifth ribs on the left side posteriorly as well as of the bodies of the twelfth dorsal and the first lumbar vertebrae (Fig. 33 A).

Under fluoroscopic control a small catheter was passed through the esophagus into the stomach without meeting with any obstruction. The catheter was withdrawn to the junction of the upper and the middle thirds of the esophagus and Lipiodol which was injected outlined the entire esophagus indicating its structural integrity (Fig. 33 B). At a later examination 2 cc of Diodrast was injected which promptly outlined the tracheobronchial tree. A small amount of the opaque material also entered the distal end of the esophagus and the stomach (Fig. 33 C). It is interesting to note that when the identical procedure was employed using Lipiodol instead of Diodrast the fistulous communication could not be demonstrated. It is possible that in this case the watery Diodrast was able to make its way through the fistulous communication between the esophagus and the trachea more readily than the oily Lipiodol.

The child's condition became rapidly worse and death ensued 72 hours after admission to the hospital.

The essential findings at autopsy were

1. An interventricular septum defect measuring 5×8 mm.
2. Aspiration pneumonia with hemorrhages in the lung.
3. Tracheo-esophageal fistula with a normal esophagus. It is also of interest that 27 cm from the ligament of Treitz there was a diverticulum 4 cm in length with a normal mucosal lining that opened freely into the intestine. Figure 33 D shows the appearance of the fistulous opening on the esophageal side. Figure 33 E shows the opening of the fistula from the tracheal side.

CONGENITAL NARROWING OF ESOPHAGUS

Narrowing of the esophagus other than

that due to actual atresia may also be of congenital origin

Roentgenologically, the esophagus in this condition does not show the blind-sac type of termination noted in actual atresia. Although the esophagus is narrowed, the lumen remains sufficiently wide so that nutrition is not seriously interfered with. There is no evidence of an esophageal-tracheal fistula. In the cases I have seen the narrowing involved the distal portion of the esophagus for approximately a third of its length. The contour was entirely smooth.

The diagnosis of congenital stenosis of the esophagus other than atresia depends, therefore, on the following triad

- 1 Roentgen evidence of a diffuse narrowing of the distal portion of the esophagus
- 2 Smoothness of outline of this stenosed area
- 3 A history of difficulty in swallowing since birth

Illustrative Cases Congenital narrowing of the esophagus compatible with life and a fair degree of health is illustrated by the following cases

J T, female, aged 7 years. When this child was 3 days old, she began to have projectile vomiting after every feeding. The formula was changed repeatedly, to no avail. At 1 year, she weighed only 13 pounds. She was hospitalized repeatedly for long periods without any improvement. In the past 1½ years she had been having rather severe abdominal pain. Esophagoscopy revealed an ulcerated area, which bled easily. Repeated examinations showed multiple ulcers in and about the cardia, with stricture of the distal esophagus. This was successfully dilated. Further esophagoscopy at a later period showed a widening and a pouch formation 22 cm from the teeth, with an area of constriction just distal to it. Dilatation of the areas of constriction was carried out through the esophagoscope, with improvement. The roentgen examination (Fig 34) revealed a smooth narrowing of the distal half of the esophagus. Because of the onset of symptoms at birth, this may justifiably be considered a case of esophageal stricture of congenital origin.

Another example of narrowing of the esophagus of congenital origin is illustrated by the following case

R W, male, aged 3 years. This was the first admission to the hospital of this 3-year-old boy with progressive marasmus and



FIG 34 Congenital narrowing of the esophagus



FIG 35 (A, *Left*) Congenital narrowing of the esophagus (B, *Right*) Same case Appearance after instrumental dilatation

anemia. The infant was a full term $7\frac{1}{2}$ pound baby, delivered by forceps with no apparent birth injury. From birth, the infant did not take his formula well and gained weight slowly. Development up to 6 months did not appear to be much retarded, as he had his first tooth and could sit up. He stood at 11 months but never walked or talked and seemed to have made little progress after the first 6 months. At the time of admission, he weighed only 16 pounds 9 ounces, vomited frequently and refused solid foods. The mother always had to dilute such foods as bananas, mashed potato and pureed vegetables before the baby would take them. Six months before admission, the child refused everything but milk and cereal. The mother definitely denied that the child had ever swallowed a caustic.

On examination he was found to be a veritable bag of bones very irritable, with marked pallor.

A gastrostomy was done, following which the child improved. Irrigations of the esophagus were done frequently.

Two months after the gastrostomy, esophagoscopy showed a tight stricture at the distal end, admitting only a Number 10 bougie. There was no edema or granulation at the site of stricture. The stricture was dilated repeatedly through the esophagoscope with success and the patient was discharged considerably improved. The child was mentally defective.

Roentgen examination of the esophagus prior to the gastrostomy (Fig 35 A) revealed almost complete obstruction at the junction of the lower and middle thirds, with marked dilatation proximal to this narrowed region. A trace of barium was noted passing through the distal portion of the esophagus. It was my opinion that the findings could be best explained on the basis of a congenital narrowing and that the almost total constriction at this time might be due to superimposed obstruction caused by retained food.

The roentgen examination after the stricture had been dilated (Fig 35 B) showed a marked diminution in the degree of constric-



FIG 36 (R T) Congenital narrowing of the esophagus. Note the smooth constriction of the distal segment

tion, although considerable narrowing was still present

The smooth constriction of the distal portion of the esophagus of congenital origin is further illustrated by the case of the patient R T male, aged 13. Since birth he had difficulty in swallowing. It was at first ascribed to some fault in the mother's milk, and the patient was weaned on the second day of life. After that the patient could swallow only liquids and such foods as custards and chocolate pudding.

Attempts were made to x-ray the child, but no radiographic examination was actually made until the child was 11 years old.

At that time a diagnosis was made of congenital stricture of the esophagus. Since that time the esophagus had to be dilated by bougies about 16 or 17 times. After each dilatation, the patient was somewhat better for about two weeks, so that bits of potato and bread could be swallowed. Despite dilatations, the patient believed that recently his condition had become slightly worse. If the patient attempted to swallow food that was not liquid, he either regurgitated it or it stuck in his throat, sometimes for days, until regurgitated.

Physical examination revealed a 13-year-old white boy, exhibiting no external evidence of abnormality. The child appeared to be undernourished, but otherwise his general condition was good.

At the time of admission to the hospital the patient was able to swallow milk, soft puddings and fluids. Repeated dilatations of the esophagus were done with bougies up to No. 32.

Radiographic examination (Fig. 36) revealed the presence of a diffuse narrowing of the distal third of the esophagus. The contour throughout was quite smooth. There was moderate dilatation above the region of narrowing. An irregularly outlined translucent area at the uppermost portion of the narrowed distal third of the esophagus probably was due to food retention. In view of the history of difficulty in swallowing with regurgitation since birth, this is evidently a case of benign stricture of congenital origin.

REFERENCES

- 1 Bayford, D. An account of a singular case of obstructed deglutition, *Mem Med Soc*, London 2 275, 1794.
- 2 Quain, Richard. The Anatomy of the Arteries of the Human Body, vol I, plate XX, Fig 7, 1844.
- 3 Holzapfel, Gotthold. Ungewöhnlicher Ursprung und Verlauf der Arteria Subclavia Dextra, *Anatomische Hefte* Referate und Beiträge zur Anatomie und Entwicklungs-Geschichte 12 373-523, Wiesbaden, J. J. Bergmann, 1899.
- 4 Harvey, R. H. Notes on two cases of anomalous right subclavian artery, *Anat Rec* 12 329, 1917.
- 5 Kirby, John. A case in which suffocation was produced by a portion of solid food in the oesophagus, *Dublin Hospital Re-*

- ports and Communications in Medicine and Surgery 2 224, 1818
- 6 Batten, Fred E A case of "dysphagia lusoria," *Lancet* 1 1579, 1895
 - 7 Brean, Henry P, and Neubauser, E B D Syndrome of aberrant right subclavian artery with patent ductus arteriosus, *Am J Roentgenol* 58 708, 1947
 - 8 Shellshear, J L and Anderson J Oesophageal atresia associated with an abnormal right subclavian artery, *Chinese M J* 41 103, 1927
 - 9 Kommerell, B Verlagerung des oesophagus durch eine abnorm verlaufende Arteria Subclavia Dextra (Arteria lusoria) *Fortschr Geb Rontgenstrahlen* 54 590 1936
 - 10 Copleman, B Anomalous right subclavian artery, *Am J Roentgenol* 54 270, 1945
 - 11 Neubauser, E B D Roentgen diagnosis of double aortic arch and other anomalies of the great vessels, *Am J Roentgenol* 56 1, 1946
 - 12 Felson, B Cohen, S Courter, S R and McGure, J Anomalous right subclavian artery, *Radiology* 54 340, 1950
 - 13 Poynter, C W M Arterial anomalies pertaining to the aortic arches and the branches arising from them, *University Studies, Univ of Nebraska* 16 229, 1916
 - 14 Combes R H and Christopherson, C Transposition of the aortic arch, *St Barth Hosp Rep* 20 273, 1884
 - 15 Herringham W P An account of a case where a right aortic arch passed behind the oesophagus to the left side and becoming dilated killed the patient by slow compression of the trachea *Tr Clin Soc London* 25 46, 1892
 - 16 Dittrich P Über einigen Variantenbildungen im Bereiche des arcus aortae *Ztschr f Heilk* 7 65, 1886
 - 17 Mohr Zur Diagnostik der kongenitalen Herzfehler *Deutsche Ztschr Nerven* 47 271 1913
 - 18 Arkin A Totale Persistenz des rechten Aortenbogens im Rontgenbild, *Wien Arch f inn Med* 12 385 1926
 - 19 Renander A Roentgen diagnosed anomaly of esophagus and arcus aortae—dysphagia lusoria, *Acta radiol* 7 298 1926
 - 20 Faber, H K, Hope J W and Robinson I L Chronic stridor in early life due to persistent right aortic arch *Acta radiol* 7 298 1926
 - 21 Loweneck M Einige seltene Beobachtungen aus der Oesophaguspathologie, *Fortschr Geb Rontgenstrahlen* 35 1230, 1927
 - 22 Biedermann F Der rechte Aortenbogen im Rontgenbild, *Fortschr Geb Rontgenstrahlen* 43 168, 1931
 - 23 Blackford L M Davenport T F, and Bayley R H Right aortic arch, *Am J Dis Child* 44 823, 1932
 - 24 Bedford, D Even, and Parkinson J Right sided aortic arch, *Brit J Radiol* 9 776, 1936
 - 25 Fray, W W Right aortic arch, *Radiology* 26 27 1936
 - 26 Robb, G P, and Steinberg I Visualization of the chambers of the heart, the pulmonary circulation and the great blood vessels in man, a practical method, *Am J Roentgenol* 41 1, 1939
 - 27 Shaw, D L An aorta with a double arch *JAMA* 28 528 1897
 - 28 Snelling C E, and Erb I H Double aortic arch, *Arch Dis Childhood* 8 401, 1933
 - 29 Gross, R E, and Ware, P F The surgical significance of aortic arch anomalies *Surg, Gynec & Obst* 83 435 1946
 - 30 Wolman, I J Syndrome of constricting double aortic arch in infancy *J Pediat* 14 527, 1939
 - 31 Neubauser, E B D Tracheo esophageal constriction produced by right aortic arch and left ligamentum arteriosum, *Am J Roentgenol* 62 493, 1949
 - 32 Paul R N A new anomaly of the aorta Left aortic arch with right descending aorta, *J Pediat* 32 19, 1948
 - 33 Edwards, J E Retroesophageal segment of the left aortic arch right ligamentum arteriosum and right descending aorta causing a congenital vascular ring about the trachea and esophagus, *Proc Staff Meet, Mayo Clin* 23 108 1948
 - 34 Wurtz, K G and Powell N B Two unusual vascular and cardiac anomalies 1 vascular ring of esophagus and trachea with patent ductus arteriosus origin of left subclavian and carotid arteries 2 persistent atrioventricular communis and aortic dextroposition with mongolism, *J Pediat* 33 722 1948
 - 35 Carson Merl J, and Goodfriend, J Constricting vascular rings *J Pediat* 34 155 1949
 - 36 Ballantyne J W Manual of Antenatal Pathology and Hygiene, Baltimore, Wood vol 2, p 462 1905
 - 37 Billard, C Traite des maladies des en

- fants nouveaux nés et à la mamelle, p 271, Paris, Baillière, 1828
- 38 Blásius, G *Observationes Medicae Rariores*, p 53, Observation 8, Amsterdam, Abraham Wolfgang, 1677
 - 39 Habershon, S O *Pathological and Practical Observation on Diseases of the Alimentary Canal, Esophagus, Stomach, Cecum and Intestines*, p. 145, London, Churchill, 1857
 - 40 Gjørup, Ernst Un cas d'oesophage double et estomac double, *Acta paediat* 15 90, 1933
 - 41 Butler, C L, and Ende, M Double esophagus with carcinoma in one Report of a case with autopsy, *Arch Path* 49 605, 1950
 - 42 Mackenzie, Morrell *Diseases of the Esophagus, Nose and Nasal Pharynx*, p 219, 1884
 - 43 Rossi *Memorie dell'Accademia delle Scienze di Torino*, v 30, series 1a, p 155, 1826
 - 44 Rossi *Bolletino delle Scienze Mediche di Torino*, v 19, p 267, 1851
 - 45 Clark, J Payson Congenital web of oesophagus—report of a case, *Tr Am Laryng A* 33 187, 1911
 - 46 Beatty, C C Congenital stenosis of the esophagus, *Brit J Dis Child* 25 237, 1928
 - 47 Abel, A Lawrence Esophageal Obstruction, p 50-57, London, H Milford, 1929
 - 48 Guthrie, K J Congenital malformations of the oesophagus, *J Path & Bact* 57 363, 1945
 - 49 Plass, E D Congenital atresia of the esophagus with tracheo-esophageal fistula associated with fused kidney, *Johns Hopkins Hosp Rep* 18 259, 1919
 - 50 Ysander, Fredrik Zur Frage der Genese der Oesophagusatresien, *Upsala Lakareforenings Forhandlingar*, N Y. Foljd 30 195, 1924-1925
 - 51 Gruenwald, Peter A case of atresia of the esophagus combined with tracheo-esophageal fistula in a 9 mm human embryo and its embryological explanation, *Anat Rec* 78 193, 1940
 - 52 Gibson, Thomas *The Anatomy of Humane Bodies Epitomized wherein All the Parts of Man's Body, with their Actions and Uses, are Succinctly Described According to the Newest Doctrine of the most Accurate and Learned Modern Anatomists*, London, Awnsham & John Churchill, 1703
 - 53 Martin, M. *Société royale de méd. de Marseille*, p 44, 1820
 - 54 Houston, John *Pathological Observations*, *Dublin Hospital Reports and Communications in Medicine and Surgery* 5 310, 1830
 - 55 Andral, G *Précis d'anatomie pathologique*, v II, part I, p 271-274, Paris, Gabon, 1829
 - 56 Vrolik, W *Tabulae ad Illustrandam Embryogenesin Hominis et Mammalium, tam Naturalem quam abnormem*, Tabula 89, Fig 2 & 3, Amstelodami, G M P Londonck, 1849
 - 57 Keith, A Constrictions and occlusions of the alimentary tract of congenital or obscure origin, *Brit M J* 1 301, 1910.
 - 58 Haight, Cameron, and Towsley, Harry A Congenital atresia of the esophagus with tracheo-esophageal fistula, *Surg, Gynec & Obst.* 76 672, 1943
 - 59 Lamb, D S A fatal case of congenital tracheo-esophageal fistula, *Philadelphia Med Times*, pp 704-707, Aug 9, 1873
 - 60 Pinard, M Vices de conformation—absence d'anus, communication de l'intestin et de l'urethre fistule tracheo-esophagienne, *Bull Soc anat Paris* 48 682, 1873
 - 61 Imperatori, C J Congenital tracheo-esophageal fistula, without atresia of the esophagus Report of a case with plastic closure and cure, *Arch Otolaryng* 30 352, 1939
 - 62 Gegenbach, F P, and Dobos, E I Congenital tracheoesophageal fistula (2 verified cases and 1 presumptive), *J Pediat* 19 644, 1941
 - 63 Leven, N L, and Lannin, B G Congenital atresia and congenital tracheo-esophageal fistula, *Journal-Lancet* 65 179, 1945
 - 64 Haight, Cameron Congenital tracheo-esophageal fistula without esophageal atresia, *J Thoracic Surg* 17 600, 1948
 - 65 Swenson, Orvar Diagnosis and treatment of atresia of the esophagus and tracheo-esophageal fistula, *Pediatrics* 1 195, 1948
 - 66 Abbott, O S, and Hopkins, W A Congenital esophageal atresia and tracheo-esophageal fistula, *J M A Georgia* 40 44, 1951
 - 67 Cardulo, H M, and Berens, D L Tracheoesophageal fistula unassociated with atresia or stenosis, *New England J Med* 243 853, 1950.

Displacement and Compression of the Esophagus by Extra-esophageal Lesions

CARDIAC DISEASE

Marked dilatation of the left auricle causes a displacement in the position of the esophagus posteriorly. As a rule, such displacement does not produce sufficient compression to give rise to actual clinical manifestations of dysphagia, but in one case the difficulty in swallowing was so marked that a clinical diagnosis of carcinoma of the esophagus was made. However, prior to operation, fluoroscopic study showed the cause to be marked compression of the esophagus by an extremely enlarged heart. The patient was treated medically for her cardiac condition with prompt alleviation of her dysphagia.

The first studies on the relation of the position of the esophagus in cardiac enlargement were those of Kovacs and Stoerck,¹ Gabert and Rosler and Weiss.³

Rigler,⁴ in this country, showed the change in position of the esophagus produced by abnormalities of the heart and the aorta. Brown and McCarthy⁵ made a comprehensive study of alterations from the normal position of the esophagus produced by various conditions such as thoracic deformities, pleuropulmonary affections, mitral valve lesions, aortic valve lesions, general enlargement of the heart, congenital heart disease, dilatation of the aorta and pericarditis.

In the presence of mitral valvular disease the esophagus is displaced to the right and posteriorly. The explanation for the displacement to the right is that there is a rotation of the heart due to enlargement

of the right ventricle. The posterior displacement is a direct result of the pressure exerted by the enlarged left auricle. The enlarged left ventricle may also participate. When both the left auricle and the left ventricle are enlarged, the displacement may affect the entire course of the esophagus in relation to the heart. Pericardial effusion produces only slight displacement of the esophagus. Cardiac enlargement in the presence of lesions of the aortic valve displaces the esophagus to the left and backward.

Schwedel and Gutmann⁶ compared the roentgenologic findings with those at autopsy by means of a life size model made of molding clay. They demonstrated the exact relationship of the heart and the aorta to the displacement of the esophagus in the roentgenogram.

Kyphoscoliosis may cause considerable change in the position of the upper and the middle portions of the esophagus. Other causes of external origin which may produce considerable displacement and compression of the esophagus are aneurysm of the aorta and tumors and abscesses of the lung and mediastinal lymph nodes.

Illustrative Case. The degree to which displacement of the esophagus may occur as the result of cardiac enlargement is illustrated in Figure 37.

This patient in addition to clinical evidence of serious cardiac disease had complained of difficulty in swallowing for a period of 2 years. In this case the posterior displacement of the esophagus (Fig 37 A) was produced by a massive left auricle (Fig 37 B). The findings were confirmed at autopsy.

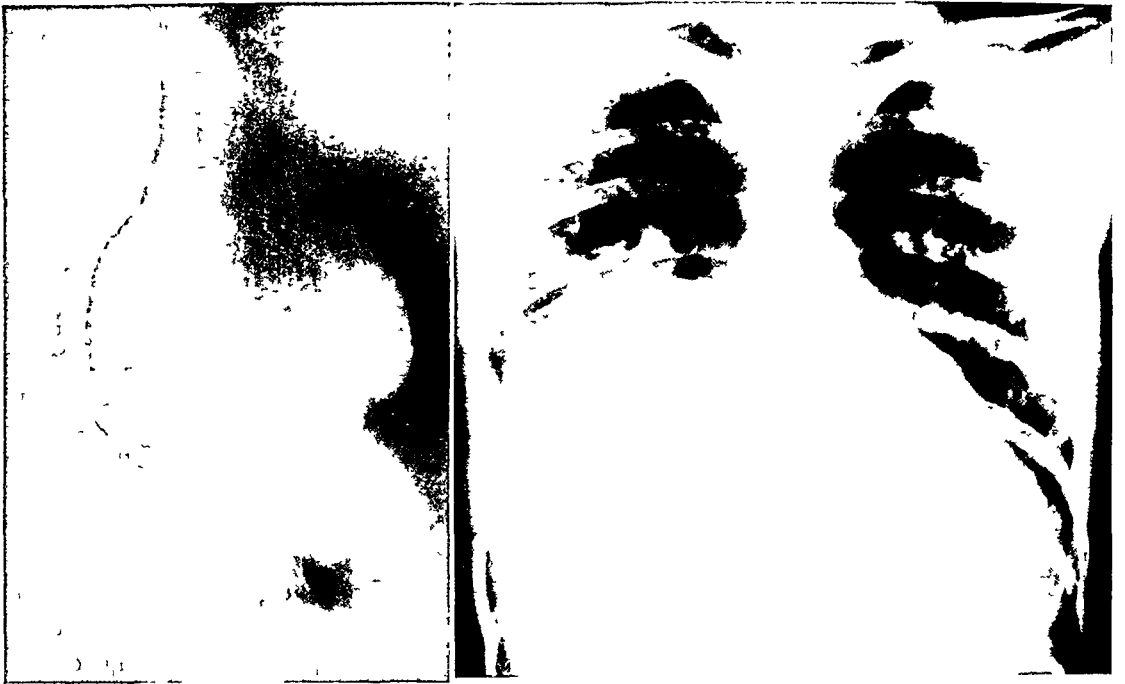


FIG 37 (A, *Left*) Marked displacement of the esophagus by a massive left auricle (B, *Right*) Massive left auricle in the same patient

AORTIC ANEURYSM

of this are well illustrated by the following case

Illustrative Case C P, male, aged 47 years This patient had a history of chancre at

Compression and displacement of the esophagus may occur as a result of aneurysm of the aorta The roentgen characteristics



FIG 38 A Huge aneurysm of the descending aorta



FIG 38 B Same patient as shown in Figure 38 A. Marked anterior displacement with compression of the esophagus by an aneurysm of the descending aorta.



FIG 39 Note the posterior displacement of the proximal portion of the esophagus when the aneurysm involves the aortic arch.

the age of 15. He was well until 15 months before his death. His main complaint was a severe radiating, knifelike pain in the left chest posteriorly and in the lower thoracic spine. A nerve block was done to alleviate pain. Toward the end the patient complained of difficulty in swallowing.

Roentgen examination (Fig 38 A) revealed a huge aneurysm of the descending aorta at the level of the seventh to the tenth dorsal vertebra. There was considerable anterior displacement with compression of the esophagus (Fig 38 B).

Autopsy revealed an aneurysm of the descending aorta. The aneurysm measured 6 inches transversely, 6 inches in its longitudinal axis and about the same in its anteroposterior extent. The mass occupied the major portion of the posterior part of the thorax from the left posterior axillary line to the angle of the ribs on the right. The esophagus occupied its normal position above the mass, then curved out forward and to the right over the aneurysm, returning to its normal position at the diaphragmatic hiatus. There was erosion of the ribs and of the mid dorsal vertebrae.

Figure 39 shows the posterior displacement of the esophagus when the aneurysm



FIG 40 Note the anterior displacement and narrowing of the lumen of the distal portion of the esophagus by the aneurysm of the descending thoracic aorta (confirmed operatively).

involves the arch of the aorta. This is in contrast with the forward displacement of the esophagus when the aneurysm arises distal to the arch.

The distal segment of the esophagus may be displaced anteriorly and somewhat compressed by an aneurysm of the descending portion of the aorta as demonstrated by this operatively confirmed case (W. W., male, aged 55). Nine months before admission to the hospital the patient began complaining of epigastric knifelike pain radiating to the back, it came on almost immediately after eating and persisted until it was relieved by vomiting. He had lost 48 pounds during this period. There was a history of a penile lesion at the age of 25. His Wassermann was 4+. Radiographic examination revealed an aneurysm of the distal portion of the descending thoracic aorta. The diagnosis was confirmed by kymographic cardiography and aortography.

Figure 40 shows the marked forward displacement of the distal portion of the esophagus, as well as the narrowing of the lumen of this segment by the aneurysm.

Operative findings "At about the level of the ninth thoracic vertebra there was a saccular aneurysm of the descending aorta. A small portion of the aneurysm pointed laterally to the left. The larger portion, measuring 5 cms., pointed medially and anteriorly, displacing the esophagus, and in the upper border the aneurysm was firmly adherent to the wall of the esophagus. The wall of the aneurysm was thin and compressible, and the entire aneurysmal sac pulsated."

The aneurysm was freed, and an attempt was made to dissect it away from its attachment to the esophagus, except at the upper border, where it could not be separated from the esophageal wall. A trocar was placed into the aneurysm, and about 4½ feet of magnesium wire was threaded into the sac. The patient made a good clinical recovery which, however, was only temporary, the symptoms recurred a few months later.

EXTRA-ESOPHAGEAL TUMOR

Posterior displacement of the esophagus by a carcinoma of the bronchus is illustrated by the following case.

Illustrative Case R. C., male, aged 49. During the 2 weeks before admission he had become increasingly weak, short of breath and hoarse and had complained of cough, of pain and discomfort in the anterior chest. He also had difficulty in swallowing solid but not soft food or fluids. Physical examination of the neck disclosed a mass of posterior cervical nodes on the left side just behind the lower end of the sternomastoid. They were very hard but not fixed or tender.

Autopsy disclosed a bronchogenic carcinoma (oat-cell type) of the right main bronchus with metastases to mediastinal and cervical lymph nodes, lungs, right perirenal tissues and right kidney, and compression of trachea, bronchi, esophagus and great vessels of the mediastinum and the neck. The esophagus throughout its course was free of any infiltration.

Roentgen examination of the chest (Fig. 41 A) showed evidence of a pulmonary neoplasm. Examination of the esophagus (Fig. 41 B) revealed considerable narrowing near the junction of the lower and middle thirds. The esophagus was displaced posteriorly in this region, and the contour of this narrowed area was essentially smooth. The narrowing and posterior displacement of the esophagus were considered to be of extra-esophageal origin, produced by the pressure of the bronchogenic tumor.

Stenosis and irregularity of the contour of the esophagus may be caused by metastatic nodes from a bronchogenic carcinoma.

Illustrative Case A. Z., male, aged 45. Two months before admission to the hospital the patient developed difficulty in swallowing solid foods but was able to take fluids. During the preceding months he also had difficulty in talking and spoke in a husky whisper. Also during the past few months he had a chronic cough, with sputum that recently had been blood-streaked. He had night sweats, extreme weakness, weight loss and some precordial burning pain, which was almost constant and radiated to the left shoulder. He had imbibed from 1 to 1½ quarts of whisky a day before the present episode. He had lost 20 pounds in the preceding 2 months.



FIG 41 A Tumor of the lung—bronchogenic carcinoma



FIG 41 B Same case as is shown in Figure 41 A Compression of the esophagus by the tumor

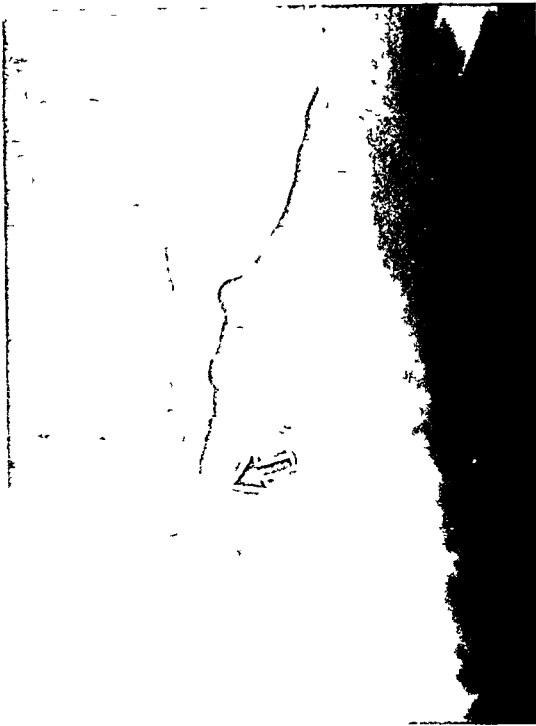


FIG 42 Constriction of the esophagus by metastatic nodes from carcinoma of the lung



FIG 43 Marked narrowing of the esophagus by an extra-esophageal tumor

Physical examination showed a chronically ill, emaciated individual. Numerous nodes were palpable in the neck and, in particular, one firm, hard node in the left supraclavicular region, not freely movable.

Esophagoscopy showed no evidence of any intrinsic organic disease.

Bronchoscopy showed no evidence of tumor masses, although the chest service believed that a bronchogenic carcinoma was present on the basis of the roentgen examination of the chest.

Autopsy revealed an adenocarcinoma of the right lower lobe of the lung with secondary carcinoma of all lobes of the lung. There was secondary carcinoma in the tracheo-bronchial lymph nodes with extension into the adventitia of the esophagus, with marked compression stenosis of the esophagus. The autopsy report read as follows: "The esophageal mucosa is smooth throughout. There is a constriction of the esophagus 18 cm from its origin. The esophagus is dilated slightly for 3 cm above this point of constriction. At the point of constriction the lumen of the esophagus is reduced to 1 cm. The constriction is smooth. At 15 cm from its origin on its posterior surface, there is a small polyp which measures 6 cm in diameter. It is not ulcer-

ated. Its borders are sharp and clearly defined. The remaining portion of the esophageal mucosa is completely smooth."

Roentgen examination of the esophagus (Fig 42) showed an area of constriction at about the junction of the upper and the middle thirds. There was moderate dilatation of the esophagus above this region. Just distal to the smoothly outlined portion the esophagus showed irregularity of contour, with varying gradations of constriction. This narrowed area gradually faded into the more smoothly outlined portions of the esophagus, both proximal and distal. The possibility of a new growth of the esophagus was considered. As disclosed at autopsy the stenosis and irregularity in the configuration of the esophagus were due to compression by metastatic nodes from the carcinoma of the lung.

An extra-esophageal tumor as a cause of a smooth constriction of the esophagus is illustrated by the following case:

Illustrative Case E J, female, aged 29. The patient complained of a lump of 3 weeks' duration on the left side of her neck. There was a moderate, unproductive cough. Physical examination revealed a firm, nontender mass

from about 3 to 4 cm in diameter, not attached to the overlying skin. In addition, there were several small shotty lymph nodes. The biopsy report was "reticulum cell lympho sarcoma." The patient had x-ray treatments, with disappearance of the nodes. She then developed a lump in the left axilla. She lost 10 pounds in weight. She had knifelike pains in her chest. Two months prior to admission to the hospital she noticed that she could not swallow solid food but could swallow milk and soups. She had pain and coughed when she tried to swallow solid foods.

Esophagoscopy examination showed a definite narrowing 28 cm from the upper teeth. A No 18 French bougie could be passed through the stricture. The upper end of the stricture was approximately 10 cm from the stomach. The stomach was entered with the bougie, following which there was a gush of gas from the stomach. The esophagus proximal to the stricture was normal.

The essential findings at autopsy, as far as they relate to the esophageal condition were:

Immediately below the bifurcation of the trachea in the left main bronchus, a grayish white, hard tumor nodule about 2 cm by 1 cm projects slightly into the lumen of the bronchus. It extends posteriorly and is closely adherent to the esophagus. The hilar lymph nodes are enlarged, firm and oval and are converted into homogeneous gray white tumor

masses, as are the mediastinal lymph nodes.

"At a point at the level of the bifurcation of the trachea, the lumen of the esophagus narrows suddenly. Nothing larger than the tongs of a regulation blunt forceps passes the level of the stricture. The stricture, which extends for about 1 cm, is the result of a complete collarlike encirclement of the esophagus by the tumor nodule described at the tracheal bifurcation. The esophageal mucosa is somewhat injected immediately above the stricture but is otherwise normal."

Microscopic examination revealed the presence of a primary bronchogenic carcinoma of the left main bronchus.

Roentgenographic examination of the esophagus (Fig 43) showed a marked narrowing at about the level of the bifurcation of the trachea. The outline of this narrowed region was very sharply defined and was perfectly smooth. There was moderate dilatation of the esophagus above this region. The extremely smooth character of the outline suggested that the constricting lesion was probably benign.

A carcinoma of the lung may produce anterior displacement of the distal portion of the esophagus with secondary narrowing of the lumen and dysphagia, as illustrated by the following case:



FIG 44 Anterior displacement and constriction of the distal segment of the esophagus by a carcinoma of the lung. (A Left) Carcinoma of the right lung. (B Right) Note the forward displacement and constriction of the lumen of the esophagus produced by the tumor accounting for the dysphagia.



FIG 45 Carcinoma of the right upper lobe of the lung with secondary distortion of the contour of the proximal portion of the esophagus

W B, male, aged 70. This patient was admitted with a history of progressive dysphagia of 6 months' duration so that he was able to take only fluids by mouth. He also stated that he had sharp chest pain which was aggravated by coughing and eating. There was progressive weight loss. The exact amount could not be determined. Bronchoscopic examination revealed a red granular mass involving the lateral and the medial walls of the right main bronchus. A biopsy was taken from this mass. The pathologic report was carcinoma. Papanicolaou study of a specimen of the sputum showed conclusive evidence of an epidermoid carcinoma. The final clinical diagnosis was of a primary carcinoma of the right main bronchus with forward displacement of the esophagus and narrowing of its lumen.

Radiographic examination (Fig 44 A) shows the bronchogenic tumor of the right lung. Figure 44 B shows the forward displacement of the distal portion of the esophagus by the tumor and the narrowing of its lumen which accounted for the dysphagia.

A carcinoma of the lung may secondarily invade the wall of the esophagus, producing distortion of its contour practically indis-

tinguishable from the deformity of a primary lesion. This is illustrated by the following case.

J S, male, aged 54. This patient, a chronic alcoholic, had a mild chronic cough of 30 years' duration with small amounts of mucopurulent sputum. The cough became more marked in the preceding 5 months before admission to the hospital and was accompanied by a weight loss of 50 pounds during that time. There was no hemoptysis. Physical examination showed some atrophy in the upper half of the right thorax. This area was dull to percussion. There were rales and many rhonchi and an occasional friction rub. The liver was palpable three fingers' breadth below the right costal margin on inspiration. Radiographic examination of the chest at this time showed a homogeneous density in the right upper lobe. Bronchoscopy showed fixation of the right bronchial tree with purulent exudate from the right upper lobe. A diagnosis of right upper lobe neoplasm was made. Exploratory thoracotomy revealed an inoperable tumor of the right upper lobe. Following operation he received radiation therapy.

He later developed progressive swelling of the face and the neck, increased hoarseness and difficulty in swallowing solid food, and additional weight loss. Biopsy of a right supraclavicular node showed squamous-cell carcinoma.

Physical examination revealed a thin, chronically ill white male with evidence of marked weight loss. There was generalized swelling of the face and the neck with puffiness of the eyelids. There was slight enophthalmos, ptosis and myosis of the right eye. The trachea was deviated to the right. There was bilateral venous distention of the neck and the arms. A few hard immovable nodes were present in the right cervical and supraclavicular regions.

The final clinical diagnosis was

- 1 Squamous-cell carcinoma of the right upper lobe of the lung with extension to the mediastinum and the cervical nodes
- 2 Obstruction of the superior vena cava
- 3 Horner's syndrome on the right side
- 4 Invasion of the esophagus

Autopsy confirmed the diagnosis of a squamous-cell carcinoma of the bronchus in the right upper lobe with direct extension of the carcinoma into the neck, with involvement of the right carotid structure and invasion of the wall of the esophagus. The report of the examination of the esophagus was as

follows "The esophagus is densely adherent anteriorly to the previously described neoplastic tissue found in the mediastinum, as is the aorta and the trachea. Its mucosa, however, has not been invaded and is smooth."

Radiographic examination (Fig. 45) in addition to revealing the tumor in the right upper lobe of the lung, shows evidence of deformity of the esophagus at the junction of the upper and the middle thirds, which was considered to be due to an infiltrative lesion secondary to invasion of the primary malignant tumor of the lung. Not only did this impression conform with the clinical features

of the case but it was confirmed by the findings at autopsy.

Dysphagia may result from the pressure of protuberances of the vertebrae (Zahn⁷ Pincsohn⁸ Mosher⁹ and Iglauer¹⁰).

The vertebral pathology occurred at different levels. In Zahn's cases, as also in the case described by Pincsohn, the exostoses were in the thoracic vertebrae. In the two cases described by Mosher and the one by Iglauer the pathologic process involved the cervical vertebrae.

REFERENCES

- 1 Kovacs, F., and Stoerck, O. Über das Verhalten, des Ösophagus bei Herzvergrößerung. *Wien klin Wchnschr* 23 1471 1910.
- 2 Gabert, E. Die Lagebeziehung des Ösophagus zur dorsalen Herzfläche und ihre Veränderung durch Erweiterung des linken Vorhofs im Röntgenbilde (Zur Symptomatologie der Mitralfehler). *Fortschr Geb Röntgenstrahlen* 32 410 1924.
- 3 Rosler, H. and Weiss, K. Über die Veränderung des Ösophagusverlaufes durch den vergrößerten linken Vorhof. *Beobachtungen zu der einschlägigen Arbeit Giberts* (Fortschr Geb Röntgenstrahlen 32 410 1924). *Fortschr Geb Röntgenstrahlen* 33 717, 1925.
- 4 Rigler, L. G. The roentgenologic differentiation of lesions of the right and left heart. *Radiology* 20 463 1933. and The visualized esophagus in the diagnosis of disease of the heart and aorta. *Am J Roentgenol* 21 563 1929.
- 5 Brown, S. and McCarthy, J. E. A study of the esophagus in relation to the heart, aorta, and thoracic cage. *Radiology* 24 131, 1935.
- 6 Schwedel, J., and Gutmann, E. B. The esophagus in disease of the heart and aorta, case report with roentgen and post mortem findings. *Am J Roentgenol* 34 164 1935.
- 7 Zahn, H. Ein Fall von Abnückung der Speiseröhre durch vertebrale Ekchondrose. *München med Wchnschr* 52 1680 1905.
——— Ein zweiter Fall von Abnückung der Speiseröhre durch vertebrale Ekchondrose. *München med Wchnschr* 53 906, 1906.
- 8 Pincsohn, A. Ösophagusstenose infolge vertebraler Exostosen, *Berlin klin Wchnschr* 58 20 1921.
- 9 Mosher, H. P. Exostoses of the cervical vertebrae as a cause of difficulty in swallowing. *Laryngoscope* 36 181, 1926.
- 10 Iglauer, S. A case of dysphagia due to an osteochondroma of the cervical spine osteotomy recovery. *Ann Otol Rhin & Laryng* 47 799 1938.

Diverticula of the Esophagus

TYPES OF DIVERTICULA

Diverticula of the esophagus may be classified under the following types

1 *Pulsion diverticula*, which occur mainly on the posterior wall at the pharyngo-esophageal region

2 *Traction diverticula*, which are found most frequently on the anterior wall of the esophagus at about the level of the bifurcation of the trachea

3 *Traction-pulsion diverticula*, which represent a combination of two etiologic factors (1) the primary underlying traction produced upon the esophagus, and (2) the effect of the pressure of food within the esophagus upon this diverticulum

4 *Transient diverticula*, which are demonstrable only at times, they disappear, apparently, as a result of changes in the functional behavior of the esophagus

5 *Congenital diverticula*, which do not result from the operation of any organic cause, such as an adhesion, but are the result of a developmental anomaly

6 *Epiphrenic diverticula*.

PHARYNGO-ESOPHAGEAL DIVERTICULA

The first description of a posterior pharyngo-esophageal diverticulum with autopsy confirmation was by Ludlow in a letter which he wrote to Dr Wm Hunter in 1764, but it was not published until 1769¹ He stated in this letter

The following case appearing to me extremely singular, I am induced to send it to you, that if approved of, it may be published in the next volume of the *London Medical Observations*, etc

R D, a considerable distiller of this city, near sixty years of age, and to the last degree emaciated, some time in June last applied to me for my assistance, having not been able to pass any manner of sustenance into his stomach since the morning of the preceding day His complaint having been for a very considerable time increasing, and finding him reduced to the last extremity, I desired an immediate consultation with some other gentlemen of the faculty, which was complied with, and Mr Barret and Mr Townsend being called in, our patient gave us nearly the following account

That, about five years ago, eating some large white-heart cherries, one of the stones lodged in his throat, and, after remaining there three days, in consequence of a very violent fit of coughing, it returned, during the time of the stone's being in his throat, the inconvenience was not so great as to prevent his taking his common meals, though with difficulty, nor did he apply to any surgeon for help, being rather inclined to think that what he felt did not arise from the stone being in his throat, but from its having injured that part as it passed on to the stomach, after the return of the cherrystone, he was sensible of a considerable degree of soreness in that place, where it had lodged, which continued a great while, and some time after, but it must have been at least a year, he observed, that, an hour, and sometimes two or more, after a meal, a small part of what he had last taken down returned into his mouth, not altered in its taste, and without any sickness preceding The quantity of food returned gradually increased, and the time of its remaining within him lessened, so that sometimes, in the midst of a meal, some of his food would return so suddenly as almost to strangle him, particularly when drinking, and at last he could not take above four large spoonfuls before it began to return, which it seemed to do without any part going into the stomach, and this nearly in the same manner as in ruminating animals, constantly without pain or sickness, some-

times with, sometimes without, an eructation. That no part of his food did at last pass into his stomach, he was convinced, by measuring the quantity of what he took in, and of what he returned, whereby he found that the latter exceeded the former, as much as might be reasonably expected, from the addition of the saliva. The day after our patient's death, having received instructions from his son, I opened him in the presence of Dr Lyne and Mr Townsend. I began the opening as high as the superior part of the thyroid cartilage, and thence continued it in the usual manner, so as to expose the contents of the thorax and abdomen: then dividing the *aspera arteria* and the larger blood vessels I removed both the heart and lungs and immediately thereon we observed a considerable muscular bag passing down between the *oesophagus* and *vertebrae*. The inferior point of this bag reached some little way into the cavity of the thorax hanging quite loose and detached, and principally situated on the right side. This appearance being so singular, we carefully dissected, and cut the bag, together with the *larynx*, *pharynx*, and *oesophagus* and on the closest examination it was clear that this bag was formed by a dilatation of the entire substance of the posterior part of the *pharynx*, the uniformity and thickness of both being so exact that it was impossible to ascertain at what particular point the dilatation first began.

(Reproductions of the diverticulum accompany the article.)

A classical account of a pharyngo-oesophageal diverticulum is also to be found in the report by Sir Charles Bell in *Surgical Observations* published in 1816 under the title of "A Preternatural Bag Formed by the Membrane of the Pharynx Which Impeded the Introduction of the Bougie."

A considerable time ago a gentleman called to consult me about a disease in his throat which gave him great uneasiness and occasioned difficulty of swallowing. In the course of conversation I found his anxious hopes rested on my being able to pass a bougie into his stomach, by which he said he was always relieved of his uneasy sensations, and I could learn from him that some ineffectual attempts had been lately made.

He sat down before me rather with the confidence of a man who was to be gratified than one who was about to suffer a painful and disagreeable operation. I made many in-

effectual attempts to pass the bougie into the *oesophagus*. In carrying the point towards the back part of the *pharynx*, I found an obstruction so absolute, that I could not venture to press with force. In introducing the bougie much curved, so as to avoid the obstruction on the back part, I still found it interrupted. I at last succeeded, by giving the instrument a twist laterally which brought the point to pass by the side of the throat and move along the lateral part of the *pharynx*. I observed with some interest, that when the bougie was introduced it was not grasped nor were there any impressions on the soft wax.

Bell had an opportunity of examining the body after death.

I found a bag projecting from the lower and back part of the *pharynx* and pushed into a space betwixt the *oesophagus* and spine. The bag was not covered with muscular fibres but may be described as a hernia or protrusion of the inner coat of the *pharynx*, betwixt the strong fibres of the muscular coat.

Reflecting on former experience, it was evident that this bag was the cause of obstruction by receiving the end of the bougie. For when the instrument was directed backwards the point of it must have entered the bag and when carried in an opposite direction it must have passed into the chink of the glottis. When the bougie was directed laterally it escaped both these holes and went down into the proper canal.

An excellent reproduction of the abnormality is recorded and he states it was "formed by the inner Membrane of the Pharynx, being thrust betwixt the Muscular Fibres." A hernia of the inner coat of the *pharynx* through the fasciculi of the constrictor *pharynx*.

His explanation of the etiology of the lesion was as follows:

This gentleman whose case I have shortly related, was subject to a spasmodic difficulty of swallowing. In that state it often happens that the force of the voluntary muscles remains while they are unable to overcome the spasm in the top of the *oesophagus*. There are then repeated ineffectual *cupping* or *efforts* to swallow, which distend the *pharynx* and although by such excitement the muscular fibres acquire strength, yet their strength is like that

of the bladder when stimulated by frequent nismus, the fibres form stronger and more distinct fasciculi, betwixt which, the inner membrane is permitted to be protruded

His conception is essentially similar to the one most commonly accepted at the present time, namely, that because of lack of co-ordination in the act of swallowing the transverse portion of the cricopharyngeus muscle fails to relax, and as a result there is a gradual distention of the weak triangular space above it with the ultimate development of a herniation of the pharyngeal mucosa through this area

Some of the interesting clinical features of a pharyngo-esophageal diverticulum were emphasized by Hankel (1833), quoted by Knott. He described a case of a merchant, aged 54, who died as a result of inanition because of dysphagia. He noted that following the ingestion of food a large tumor developed on each side of the larynx which could be emptied of its contents by pressure upon it with some of the food returning into the mouth. The patient had complained of the prompt regurgitation of food after each meal. At autopsy Hankel found a diverticulum originating just beneath the inferior fibers of the pharyngeal constrictor. Because of the fixed vertebral column, he noted that the distended sac compressed the esophagus posteriorly with complete occlusion, causing dysphagia and death.

Therefore, Hankel described practically all of the essential clinical features of the disorder: the dysphagia, the prompt regurgitation of ingested food, the swelling of the neck caused by the distention of the sac, the diminution in the size of the swelling by manual pressure, forcing it partially free of its contents, and the obstruction of the esophagus caused by the mechanical pressure upon it of the distended pouch.

Coffin, in 1847,³ presented a plaster model of a specimen of the pharynx which he had obtained in the case of a woman 58 years old who died of peritonitis. There was a history of the patient's having consulted her physician because of dysphagia. At au-

topsy he noted that the pharynx ended in a cul-de-sac, forming a pouch about 10 cm in length and distended with an accumulation of vermicelli and milk which the patient had eaten shortly before death. The wall of the pouch was formed by the mucous membrane of the pharynx. The esophageal opening was a narrow orifice without induration or stricture. Coffin considered the lesion to be of congenital origin, although he gave no reason for this opinion. Cruveilhier in his discussion of the case stated his belief that the pouch could be classified as a hernial protrusion.

Ogle, in 1866,¹ also described a fully documented case with autopsy confirmation of a pouchlike dilatation of the lower part of the pharynx. The patient was a 63-year-old male with a history of dysphagia of many years' duration. Small portions of swallowed food would return to his mouth during a period of several hours. At post-mortem, at the lower end of the pharynx at a point corresponding to the lower border of the inferior constrictor muscle of the hypopharynx, there was a pouch equal in size to that of a bantam's egg. The pouch involved only the posterior and the lateral walls of the pharynx. An excellent illustration of the specimen accompanies his article.

The condition was also described by Meckel⁵ and by Zenker and Ziemssen in 1877.⁶ Because of the classical description of the condition given by these authors this type of diverticulum is frequently referred to as a Zenker diverticulum. In reality from a study of the historical background, primary credit for the original contribution to the anatomy, and an elucidation of the underlying etiology of the pharyngo-esophageal diverticulum, belongs to Ludlow.

The development of these diverticula on the posterior wall at the junction of the pharynx and the esophagus may be understood from the anatomic characteristics of this region. Between the transverse and the oblique fibers of the cricopharyngeus

muscle is a triangular area which is almost free of muscle structure. Such an area is therefore anatomically predisposed to diverticulum formation. In addition, this is the narrowest area of the pharynx, and because of the unyielding character of the anterior wall, pieces of food and foreign bodies may readily stick in this region of the posterior wall.

Thereafter, with every act of swallowing resulting from the contraction of the rest of the pharyngeal musculature, the food is pressed against this less resistant region. Thereby the mucosa may be pushed outward and gradually may become exaggerated in size until the eventual development of a formed sac in which food may remain after the act of deglutition has been completed. The contents of the sac then increase its size by their weight until it extends downward between the esophagus and the spine. In the filled state, the sac may compress the esophagus so that no food may pass through it.

The possibility of a congenital origin of the diverticulum in rare instances is suggested by the observations of Brintnall and Kridelbaugh.⁷ They reported two cases of hypopharyngeal diverticula in the newborn clinically simulating atresia of the esophagus. When the diverticulum was filled with Lipiodol it had the appearance of the upper cul de sac of an esophageal atresia, since none of the Lipiodol escaped into the esophagus. The clinical impression of esophageal atresia was supported by the history of the regurgitation of swallowed food from the time of birth. However, surgical intervention showed that the esophagus in each case was normal and that the saccular structure represented a diverticulum.

In the first case operation revealed a diverticulum 2 inches long and 1 inch in diameter which arose from the posterior midline of the pharynx above the level of the cricopharyngeus muscle and extended downward into the posterior mediastinum. The diverticulum showed a well developed muscle layer as well as the mucosa. The di-



FIG 46 Pulsion diverticulum of the esophagus. Note its location at the junction of the hypopharynx and the esophagus.

verticulum was excised, and the pharyngeal opening was repaired.

In the second case operation and autopsy showed that the esophagus was normal and that a pharyngeal diverticulum was present having its origin just above the cricopharyngeus fibers in the posterior midline of the pharynx. It was 1 cm in diameter and 2.5 cm in length and extended downward in the posterior mediastinum ending in a blind pouch behind the esophagus.

Roentgen Diagnosis. Roentgenologically this type of diverticulum may be noted high up on the posterior wall at the junction of the pharynx and the esophagus; the reason for this location has been shown in the description of the etiologic factors involved. The contour is smooth. It never has a tent shaped or triangular appearance.

such as is not uncommonly seen in the traction type of diverticulum. The obvious explanation, of course, is the etiologic factor in the origin of this condition, the cause being entirely pulsion in nature and never the result of the pull of periesophageal adhesions.

It is always single. Its size varies greatly. It may be so small as to be demonstrable only with difficulty, or it may assume a huge size so that it is noted extending downward to a considerable degree through the superior mediastinum. When large, it may produce compression of the anteriorly placed esophagus. As a result, none of the barium may be noted escaping from the diverticulum into the esophagus, or when some of the barium does escape, the esophagus is observed as being narrowed and displaced anteriorly as a result of the compression.

In the erect position, the diverticulum may be seen as sharply circumscribed, with a fluid level capped by air. Roentgen visualization of the lesion, because of its high position, may be readily accomplished in examining the patient in the direct postero-anterior view. However, study of the area is also recommended by changing the position of the patient through every degree of obliquity. When a small amount of barium is administered, the diverticulum may appear in isolated fashion. As more barium is administered, some may then be noted escaping into the esophagus, unless the diverticulum is huge in size and the compression upon the esophagus considerable.

The amount of barium administered, however, should be carefully gauged under fluoroscopic control, since an excessive amount may regurgitate up into the pharynx and so enter the trachea.

While the fully developed pharyngo-esophageal diverticulum may be readily recognized, its origin roentgenologically must be sought in the very small pouch of a transient character which may be seen in a fleeting observation during the course of

contraction of the hypopharynx only to disappear when the hypopharynx is distended. Although no one has traced the actual development of the completely formed pouch from these transient diverticula, it is reasonable to assume that in some cases at least such a diverticulum may be the precursor of the full-blown lesion. The fact that they both occur in the identical anatomic region on the posterior wall at the junction of the hypopharynx and the esophagus adds strong support to this assumption.^{8,9}

Figure 46 shows a classic example of a pulsion diverticulum on the posterior wall at the pharyngo-esophageal junction. The hypopharynx is well visualized. The posterior wall is smooth and sharply outlined above the location of the diverticulum. Anteriorly, the hypopharynx shows a superficial irregularity of contour. The hypopharynx becomes narrowed where it joins the neck of the diverticulum. Anterior to the diverticulum, the esophagus is markedly constricted. This constriction, however, does not appear to be due to the pressure of the diverticulum upon it. Such areas of constriction are frequently noted in the absence of a diverticulum. This area of constriction is the result of the hyperactivity of the cricopharyngeus muscle, which plays an etiologic role in the ultimate development of the diverticulum, emerging as it does above the region of the transverse fibers of this muscle.

An operatively controlled example of a pharyngeal diverticulum is illustrated by the following case.

Illustrative Case T. S., male, aged 34. Fourteen months before his admission to the hospital, the patient ate a frankfurter and 2 days later regurgitated a piece of it. From then on he regurgitated food unchanged in character and free of blood sometimes even 48 hours after eating.

During this time he also noted gurgling noises in his neck, especially after eating at night. The condition gradually became more marked and there was considerable gagging upon attempting to swallow solid food. He lost 16 pounds in weight.



FIG 47 Pulsion diverticulum of the esophagus

Physical examination of the neck showed a slight fullness on the left side just above the clavicle and posterior to the sternocleidomastoid. On palpation there was crepitation during swallowing accompanied by a gurgling noise.

Operation revealed a diverticulum at the junction of the pharynx and the esophagus. The sac was freed up to its junction with the lower portion of the pharynx and was sutured through the fundus to the sternocleidomastoid muscle close to the upper attachment.

Roentgen examination (Fig 47) showed a typical, fairly large diverticulum of the pulsion variety on the posterior wall, originating at the pharyngo-esophageal junction.

Figure 48 is an example of a pharyngeal diverticulum showing a fluid level and air bubble.

TRACTION DIVERTICULA

Traction diverticula, in contrast with pulsion diverticula, are quite common. The



FIG 48 Pharyngeal diverticulum with fluid level and air bubble

first description was that of Rokitsansky,¹⁰ who reported one in 1842. Dittich in 1851 showed the frequency of the affection. It was Rokitsansky, however, who in 1861 gave a clear description of the underlying pathology and demonstrated the pull by the cicatrization of a bronchial node upon the esophagus as the important factor in the etiology of such diverticula. Zenker described 60 cases, verifying Rokitsansky's fundamental conception.

The origin of the traction diverticulum is to be explained as follows. An inflammatory focus involves a localized area of the esophagus. As the result of scarring and contraction a pull is exerted upon this portion of the esophagus, since the inflammatory lesion is attached to less yielding structures, such as the trachea or the bronchi. The most common source of inflammation is a bronchial or tracheal lymph node, particularly at the level of the bifurcation. Because of the increased localization of lymph nodes in this region there is a special predilection of this portion of the esophagus

for the formation of the traction type of diverticula

The node may only become adherent to the outer layer of the muscular wall or it may actually invade this layer. With contraction, the muscular layer is pulled out, and the mucosa follows. In other cases the invasion may reach the mucosa. Both layers, the mucosa and the muscle, are then pulled outward. In other cases, the node becomes adherent to the mucosa through an opening in the muscular wall as the result of a periadenitis with abscess formation. In the early period of such infection the abscess may actually break through into the esophagus. After evacuation of the pus, the perforation may then be closed off. With later contraction of the cicatricial tissue the mucosa may be pulled out through this gap in the muscular wall. This also helps to explain the presence of scar formation in the depth of the mucosa of some diverticula.

Roentgen Diagnosis The earliest attempt to diagnose a diverticulum roentgenologically was made by Reitzenstein¹¹ in 1898. In a patient of his, after the administration of a bismuth suspension, the diverticulum was observed on the fluorescent screen, following which roentgenograms were taken.

Diverticula of the traction variety are limited entirely to the esophagus itself and have never been found in the pharyngo-esophageal region. Because of the preponderance of nodes on the anterior wall of the esophagus at about the level of the bifurcation of the trachea, such diverticula are to be found most commonly in this region. By the time that roentgen examination is made, these diverticula usually appear well rounded and perfectly smooth in outline. In size, they may show considerable variation, although they usually do not achieve the size sometimes noted in a pharyngeal diverticulum. In some cases the diverticulum may be tent-shaped. This apparently represents an early stage in the process of formation before it has become rounded as the

result of pressure exerted upon it from within the esophagus itself. In some cases, a calcified node may be seen in close apposition to the diverticulum, this presumably represents the etiologic factor involved in its production. I have never noted peristaltic activity in a diverticulum of this character, presumably partly because of the attenuation of the muscular structure and partly because of the destruction of the layers in the wall of the diverticulum as a result of the inflammatory cicatrizing process.

As a rule, these diverticula are single. They are rarely multiple. In 1926, I described multiple diverticula of the esophagus which had been discovered on routine roentgen examination.¹²

In some cases of multiple diverticula the various stages in the developmental process may be traced, one being small and tent-shaped while another may appear larger and rounded. The mucosal folds of the esophagus may pass through without any evidence of radiation toward the base of the diverticulum. In other cases, however, actual radiation of the folds of the esophagus may be noted up to the base of the diverticulum. The diverticulum itself is free of folds. This feature may be of roentgenologic significance when the diverticulum involves the distal portion of the esophagus. In the case of a gastric herniation, the mucosal folds of the stomach will be preserved, whereas in a large diverticulum which because of location might simulate herniation, there will be no evidence of mucosal folds.

While ordinarily in cases of traction diverticula the roentgen examination may be carried out in the first oblique position, it is nevertheless essential that the patient be turned through every degree of obliquity, since a diverticulum not visualized at one angle may be demonstrable at another.

Kjellberg¹³ showed that roentgen examination of the esophagus at a time when the diverticulum is filled with retained food may exhibit a deformity simulating that of a submucosal tumor, producing a sharply

delimited comparatively smooth indentation of the involved wall. Only re-examination at a later time, when the diverticulum is empty and therefore can be outlined by barium, will establish clearly the real nature of the underlying abnormality.

Rarely, esophageal diverticula may be associated with the presence of a hiatal hernia of the stomach. Because of the rarity of this association, however, and the comparative frequency of traction diverticula, it is my opinion that the relationship is purely coincidental and does not prove that there is necessarily an underlying congenital factor in the development of such diverticula.

Roentgen evidence of perforation of a traction diverticulum has been described by Hawes,¹⁴ Westergren and Ragnell,¹⁵ Sigora,¹⁶ Gjorge,¹⁷ and Bayer.¹⁸

TRACTION PULSION DIVERTICULA

The third type of diverticulum, according to our classification, namely, the traction pulsion diverticulum, has already been described incidentally under traction diverticula. As stated there, this type of diverticulum represents the culmination of the interplay of two factors: (1) the pull upon the esophagus produced in the process of cicatricial contraction of an inflammatory node, and (2) the superadded factor of pulsion produced by intrinsic pressure within the esophagus. Such a diverticulum, therefore, represents only an end result of the etiologic process underlying the development of traction diverticula in general. The two forces of traction and pulsion in such cases go hand in hand. The same factors in roentgen diagnosis apply here as were described for traction diverticula.

TRANSIENT DIVERTICULA

Barsony and Polgar¹⁹ considered many of the cases of multiple diverticula of a transient nature as functional and asymptomatic and due to disturbances of innervation in the wall of the esophagus. As a result, the area of involvement does not

contract. Because of intra esophageal pressure, such localized areas of involvement became distended and diverticularlike in appearance.

Evidence in support of a functional factor in the production of this phenomenon is furnished by Knight.²⁰ He used cats because the distribution of striped and unstriped muscle in their esophagi closely simulates that of man. On stimulation of the vagi or on removal of the stellate ganglia, he produced an appearance of the esophagus similar to that of the globular segmentations in man. This is illustrated by a roentgenogram of the esophagus of the cat, exhibiting these changes after the bilateral removal of the stellate ganglia.

Simon¹ suggested that the incomplete development of the musculature of the esophagus or congenital malformations of its tissues might give rise to localized areas of diminished resistance to intra esophageal pressure, leading to the picture of transient diverticulosis of the esophagus. Simon quoted Ribbert as favoring developmental anomalies as a cause of diverticula of the esophagus, and also the work of Havlicek,²¹ whose investigations in the development of reptiles and birds have given support to the theory of the congenital origin of these diverticula.

Fleischner³ made out a strong case for traction as the etiologic factor in transient diverticula of the esophagus, rejecting the conception of Barsony that they are functional in nature.

Some of his reasons were as follows:

- 1 Such transient diverticula are found as a rule in those areas in which typical traction diverticula are most apt to develop, namely, the anterior and lateral wall of the esophagus at about the level of the bifurcation of the trachea.

- 2 In some cases such transient diverticula are found in association with typical traction diverticula.

- 3 Calcified foci are found in the peritracheal lymph nodes near the base of these transient diverticula.

4. Such transient diverticula may be found in association with paresis of the recurrent laryngeal and the phrenic nerves and in cases of bronchial asthma, conditions which may be associated with a mediastinitis

5 An important point in favor of an anatomic basis for these transient diverticula is the tendency for them to recur in the same location, due allowance being made for variations in the degree of filling with barium

6 His most important evidence is his confirmation by anatomic findings at the site of these transient diverticula in five cases which were studied at autopsy

In Fleischner's opinion, therefore, practically all diverticula, including those of a transient character, are secondary to traction, except for a few cases of pulsion diverticula based on congenital predisposition

However, the question of their functional or organic origin in every case has not been clearly established

An interesting feature in the appearance of the esophagus is that of marked undulation with a tendency to segmentation involving various portions of the esophagus and, at times, occurring throughout its entire length The appearance may simulate that of small peristaltic waves

Here, too, Fleischner considered the condition as being due to the presence of multiple adhesions involving the esophageal wall. In support of this view Fleischner stated that these wavelike and segmented areas recur in the same location, even though they may show variation as to form and size These latter variations may be explained on the basis of differences of intra-esophageal pressure associated with peristaltic activity However, adhesions, fixed at various regions throughout the esophagus, may explain their constant recurrence in the same location, but similar phenomena may be noted when the esophagus is dilated, so that, in some cases at least, the appearance must be considered as being

functional Also, the circular nature of some of the segments of the esophagus seen in this phenomenon cannot be explained readily on the basis of adhesions and suggests that the etiologic factor may be functional

In mediastinitis the contour of the esophagus may be altered with the production of irregular projections due to the pull upon the wall produced by the adherent inflammatory process These alterations are qualitatively different from the encircling types of undulations of the esophagus. In this latter phenomenon, there is certainly no roentgenologic evidence of abnormal fixation of the esophagus in any one region There is no permanent narrowing of any segment or of any localized areas of dilatation of a permanent character, nor is the configuration at all similar to the usually pointed, narrow projections of a highly irregular character seen in association with mediastinitis, which remain practically constant and are hardly affected from moment to moment by the behavior of the esophagus Instead of a comparatively small localized segment of the esophagus involved in mediastinitis, the entire esophagus at times may partake of the changes noted in these recurrent, usually smoothly outlined undulations

If these changes were produced by peri-esophageal adhesions, one would have to assume first that the process involved practically the entire length of the esophagus in some cases and that the adhesions completely encompassed the wall Otherwise, only one part of the wall would be expected to partake in the changes of contour Since the rounded areas are of globular character and frequently involve the entire contour, only adhesions of a completely encircling character running throughout the length of the portion of the esophagus involved should be expected to produce an appearance of this kind.

It is difficult to believe that such a widely disseminated pathologic process would continually escape detection in the examination

of the mediastinum in all these cases of transient diverticulosis

One of the arguments in favor of adhesions as a cause of this phenomenon is the occasional association of a true diverticulum of permanent character, yet we commonly see these changes in the absence of actual traction diverticula, and they may be found in individuals who are entirely free of any associated disorder of the esophagus. Also they may be present in individuals suffering from other associated organic lesions of the esophagus, such as carcinoma. On the whole, the evidence would seem to favor a functional basis for this phenomenon associated with localized areas of lessened resistance throughout the esophagus. Mere irritability of the esophagus alone would hardly explain the constant reduplication of the findings in some individuals at the identical areas, during prolonged fluoroscopic observation or in successive roentgen exposures. Also, when the examination is repeated at a later date, the localized areas of globular configuration may again show recurrence in identical areas.

In those cases in which there is repetition of the identical pattern, one must assume that localized areas of diminished resistance exist at these particular levels, perhaps on the basis of deficiencies of muscular structure.

The localized transient alterations in the contour are not always of a globular character. They may appear serrated, or there may be a combination of serrations and wavelike ripples. At times, the esophagus may assume a corkscrew appearance. In addition to vagaries in the developmental character of the muscular structure, irritability of the esophagus of functional origin may be an important accompaniment in the exaggeration of these peculiar phenomena.

CONGENITAL DIVERTICULA

That some diverticula of the esophagus may be of congenital origin is suggested by the embryologic development of the esophagus originating from the fusion of two segments, an upper portion arising from the retropharyngeal segment of the foregut, and a lower portion arising from the pregastric

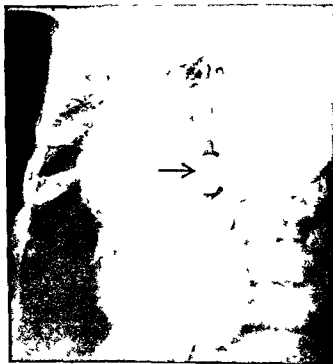


FIG 49 Diverticulum of the esophagus

segment of the foregut. A primary defect at the point of union of these two segments may be the site of the development of a diverticulum. Diverticula on the posterior wall may originate from cysts of the esophagus which are present during embryonal life. These may persist into adult life and may acquire an opening into the esophagus with the formation of diverticula. Also, as the result of imperfect separation of trachea and esophagus during embryonal life, esophageal pouches may develop.

Roentgenologically, a diverticulum may be suspected of being of congenital origin if it occurs elsewhere than on the anterior wall of the esophagus and is perfectly rounded in appearance. While the positive identification of a traction type of diverticulum is a much simpler task because of its site of predilection and its occasional tent-shaped appearance, any diagnosis as to the possible congenital origin of an esophageal diverticulum on the basis of the roentgen findings must remain entirely in the field of conjecture.

Supporting the possibility that some

esophageal diverticula may be of congenital origin is the report by Guthrie.²⁴ The case was that of a newborn female, aged 9 days, who had dysphagia from birth and turned blue on taking food. Autopsy disclosed a large, thick-walled diverticulum 1½ inches long, originating from the proximal portion of the anterior wall of the esophagus and extending downward behind the trachea. The esophagus itself was patent throughout, and there was no communication with the trachea.

O'Bannon,²⁵ also found at autopsy in a newborn a diverticulum of the esophagus at the level of the third dorsal vertebra in association with a congenital atresia.

EPIPHRENIC DIVERTICULA

A rare type of location for an esophageal diverticulum is illustrated by the epiphrenic variety. These are of smooth outline and rounded contour and in many respects have the characteristics of a pulsion diverticulum. When associated with cardiospasm, the etiologic factors underlying the development of such a diverticulum may be two-



FIG 50 (A, *Left*) Diverticulum of the esophagus, showing failure of visualization in the right oblique position (B, *Right*) Diverticulum of the esophagus visible with the patent in the left oblique position

fold (1) a weakness may be present in the wall of the esophagus above the cardiac end, and (2) exaggerated intra esophageal pressure as a result of the cardiospasm may cause a bulging of the wall of the esophagus at the site of this area of diminished resistance. Except for its rare location, the roentgen features of this type of diverticulum are essentially like those produced by pulsion elsewhere.

Illustrative Cases The following are examples of various types of diverticula of the esophagus.

T. C., male, aged 71. The patient gave a history of difficulty in swallowing for years, which had become more marked in the preceding 2 months. There had been continuous loss of weight in the preceding 1½ years.

Roentgen examination (Fig. 49) revealed a large diverticulum with a broad base, springing from the anterior wall of the esophagus. There was no evidence of infiltration, new growth, or obstruction to the flow of barium at the time of the radiographic study.

Following esophagoscopy, he developed complete obstruction, being unable to swallow even water. A gastrostomy was done.

The autopsy report read as follows: "On opening the esophagus, there is found, about 2 cm. below the region of the bifurcation, a traction diverticulum in the anterior portion of the esophagus. This diverticulum measures about 1.5 cm. in diameter and about the same in depth. It is about 10 cm. above the cardiac opening of the stomach."

The importance of examining the patient through various angles of obliquity to demonstrate the presence of a diverticulum of the esophagus is illustrated by Figure 50 A and B. In Figure 50 A the patient is in the first, or right oblique, position. There is no clear evidence of an esophageal diverticulum. Examination in the second, or left oblique position (Fig. 50 B) shows definitely a diverticulum originating from the anterior aspect of the midportion of the esophagus and directed downward. Note also that there has been regurgitation of a small amount of barium into the trachea. Clinically, the patient gave a history of coughing spells initiated by the swallowing of liquids.

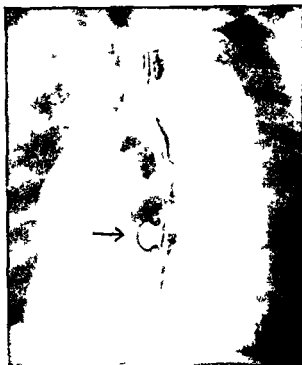


FIG. 51 Traction diverticulum of the esophagus



FIG. 52 Traction diverticulum. Note the relation to it of the calcified node

Figure 51 shows a smoothly outlined, rounded diverticulum of the anterior wall of the esophagus. The mucosal folds of the esophagus run longitudinally and do not radiate into the diverticulum. At times, however, the mucosal folds of the esophagus may be noted, radiating into the region of the diverticulum.

Figure 52 illustrates a possible etiologic relation between a diverticulum on the anterior wall of the esophagus and the presence of a calcified lymph node in close relation to it. Inflammatory pathology within the node may have been the exciting factor in the production of traction upon the esophagus with the eventual development of a diverticulum.

G. H., male, aged 50. The patient had complained of distress occurring from 10 minutes to 1 hour after meals. Roentgen examination

showed an ulcer of the first portion of the duodenum. At one time the patient complained of difficulty in swallowing food.

Roentgen examination of the esophagus (Fig. 52) revealed a cone-shaped diverticulum of the anterior wall at about its midportion. At the apex of the triangle was a calcified node, which by traction upon the esophageal wall may have been the cause of the diverticulum. The mucosal folds within the esophagus were intact.

The next case illustrates not only the presence of a definite diverticulum of the esophagus but other interesting phenomena as well. Roentgen examination revealed the fact that the diverticulum, occupying a characteristic location, was sharply pointed at its apex. This apparently represents an early stage in the developmental history of an esophageal diverticulum. Sharply pointed at first as the result of traction; eventually the contour may become rounded as the result of associated secondary pulsion in addition to the primary

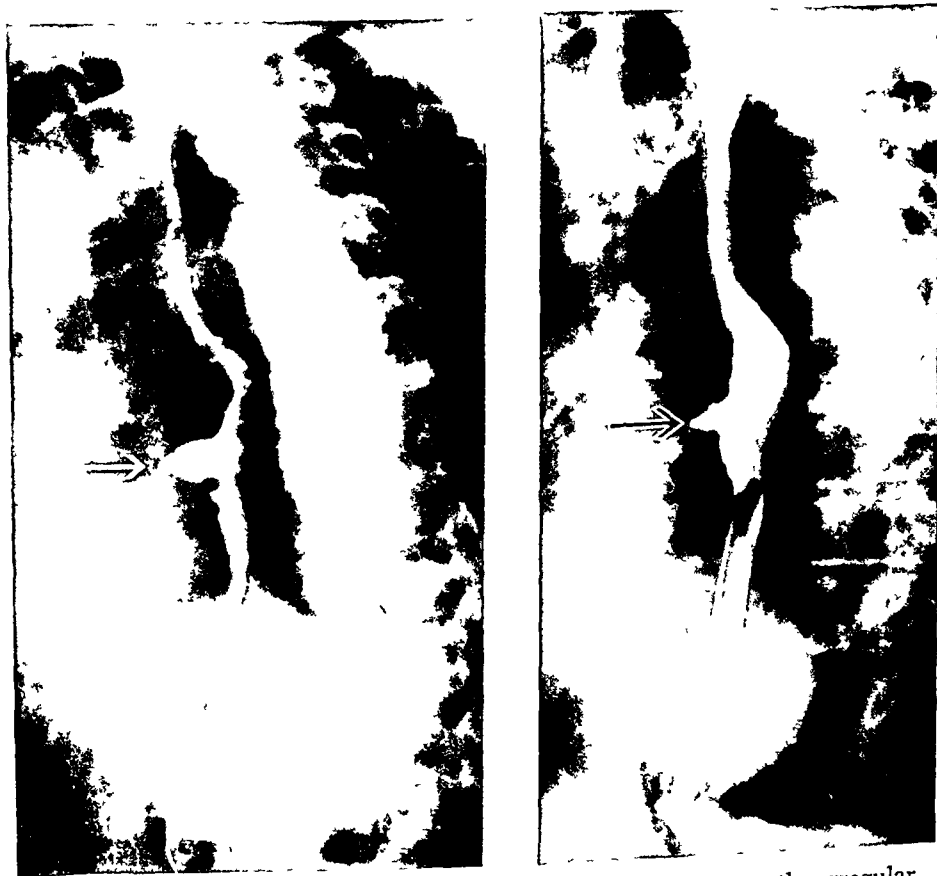


FIG. 53 (A, *Left*) Diverticulum with a conical apex. Note the irregular serrations of the esophagus. (B, *Right*) Same patient. Note the disappearance of serrations with relaxation of the esophagus.

traction. Another interesting feature of this case is the presence of numerous small projections throughout most of the esophagus (Fig 53 A). Some of these are pointed, others are more rounded in contour. That these are of a transient character is demonstrated by their absence at a time when there is complete relaxation of the esophagus, although the large diverticulum is still present (Fig 53 B).

The cause of these transient irregularities of the esophagus is probably functional irritability. This may be fundamentally similar to the phenomena of irritability present elsewhere in the alimentary tract as for example, in the stomach and the colon.

In the next case there are three diverticula originating from the anterior wall of the distal portion of the esophagus (Fig 54).

The patient (E. G.) had no symptoms referable to the esophagus. The clinical diagnosis was duodenal ulcer. In addition to the diverticula, note also the undulations of the proximal portion of the esophagus.

Rarely, a diverticulum may originate from the lower end of the esophagus, just above the diaphragm and on the posterior wall. In the following case not only does the diverticulum occupy this unusual position but it is associated with two smaller diverticula just proximal to it.

G. M. male aged 67. The patient gave a 10 year history of recurrent attacks of epigastric pain relieved by an ulcer regimen. The pain frequently woke him from sleep. There were no symptoms referable to the esophagus.

Roentgen examination showed an ulcer of the first portion of the duodenum, which explained his clinical manifestations. The findings in the examination of the esophagus were apparently incidental. The esophagus was radiographed on a number of occasions over a period of almost 5 years. Figure 55 A shows the appearance in the first, or right oblique erect position. There is a large diverticulum on the posterior wall of the esophagus a short distance above the diaphragm. The diverticulum contains an air bubble occupying the upper half. Proximal to this large diverticulum are two small, vaguely outlined saccular areas. In the second or left oblique position (Fig 55 B) the large diverticulum is

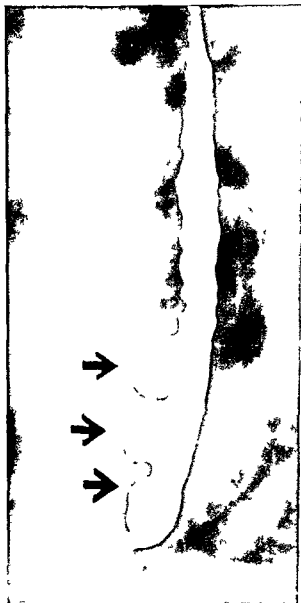


FIG 54 Multiple diverticula of the esophagus. Note also the wavelike configuration of the proximal portion of the esophagus.

seen full face. Two smaller diverticula are noted proximal to it. At the extreme distal end of the esophagus at this time is a small, rounded, saccular area. Because this was not noted in Figure 55 A and at other examinations, it was believed that it probably represented a transient ampullary dilatation of the cardiac end.

Roentgenographic examination 5 years later (Fig 55 C) showed a marked increase in the size of the diverticulum, evidently the result of pulsion.

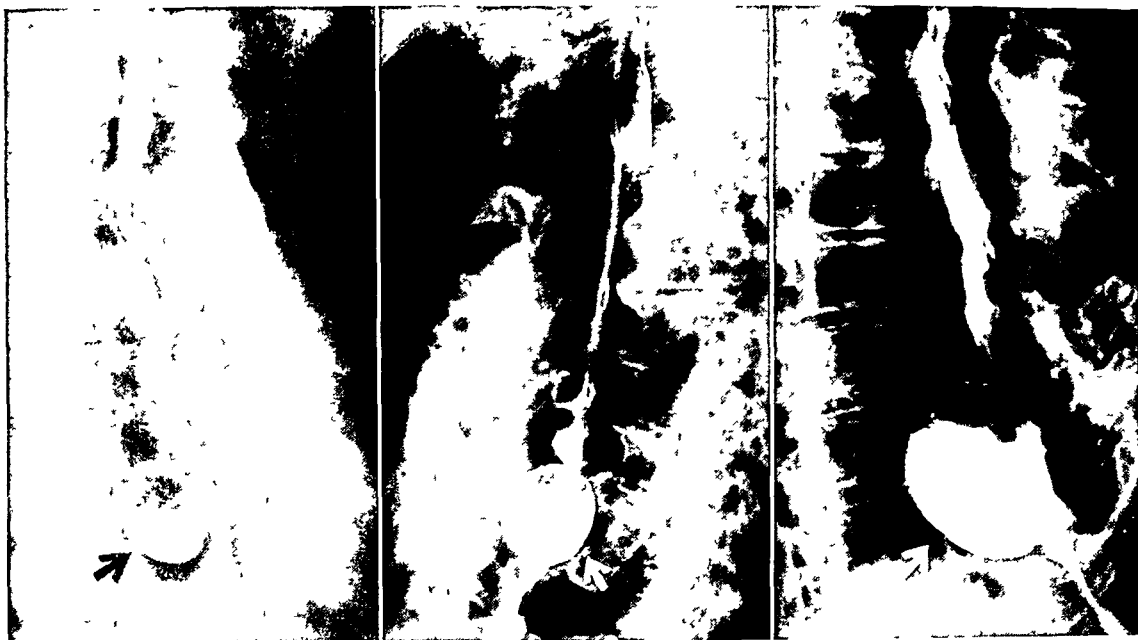


FIG 55 (A, *Left*) Diverticulum on the posterior wall of the esophagus. There are two small, vaguely outlined saccular areas proximal to this (B, *Center*) Same patient. Appearance in left oblique position (C, *Right*) Same patient. Examination 5 years later. Note the marked increase in the size of the diverticulum.

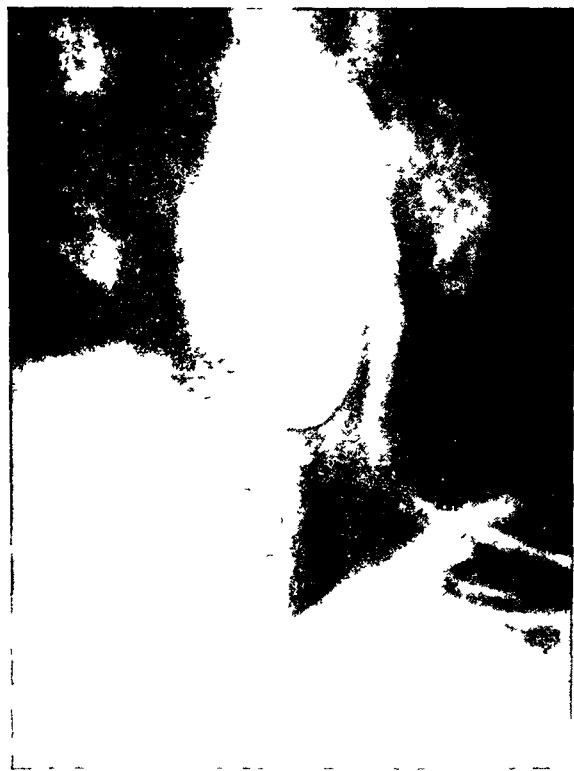


FIG 56 Large posteriorly placed epiphrenic diverticulum of the esophagus, the existence of which was corroborated at autopsy. Note the forward displacement of the segment of the esophagus from which the diverticulum originates.

A large epiphrenic diverticulum originating from the posterior wall of the distal segment of the esophagus may be noted in Figure 56. The patient (M N, male, aged 59) died, and at autopsy a carcinoma of the body of the pancreas with generalized metastatic dissemination was found. Incidentally, the diverticulum of the esophagus was also noted. The autopsy description stated: "There is a diverticulum at the lower end of the esophagus just above the diaphragm measuring 6 cm in diameter." In spite of its large size, there were no symptoms during life attributable to the diverticulum.

An unusual example of multiple diverticula of the esophagus is illustrated by the following case:

A S, female, aged 40. Four months before her admission to the hospital, she had had an obstructive sensation in her esophagus at the level of the sternum. She also complained of

occasional epigastric distress. She had had intermittent regurgitation during meals for 1 year. There was no dysphagia, anorexia or weight loss.

Physical examination was essentially negative, except for absent ankle jerks. She was under treatment for syphilis.

Because of the esophageal symptoms esophagoscopy was done. The instrument was passed without difficulty. The mucosa of the esophagus was normal throughout. About 22 centimeters from the teeth a large secondary, patent opening was found leading off from the right anterolateral wall of the esophagus. The opening appeared to be pencil size. No pathology of the mucosa was present.

The diagnosis was "diverticulum of the esophagus."

Roentgen examination (Fig. 57) revealed a rather large diverticulum on the anterior wall of the esophagus at about the level of the bifurcation of the trachea. Proximal to this area was a much smaller diverticulum also springing from the anterior wall. On the posterior wall of the esophagus at about the same level was a saccular area with a broad

base, and near its upper margin was a small square shaped projection.

Apparently, all the esophagoscopist was able to see was the opening leading to the larger diverticulum with its somewhat narrowed neck. If this structure is to be explained on the basis of traction, one must assume that there were multiple areas of adhesions, involving both the anterior and the posterior walls of the esophagus at approximately the same level. Note that on esophagoscopy there was not the slightest evidence of intraluminal organic disease and that the mucosa was entirely normal throughout.

Multiple diverticulosis involving almost the entire esophagus is illustrated by the next case.

S. Z., male, aged 64. The patient was suspected clinically of having a carcinoma of the tail of the pancreas. There was no history of dysphagia. The findings on roentgen examination of the esophagus were coincidental. At the first examination (Fig. 58 A), the esophagus showed numerous saccular areas through

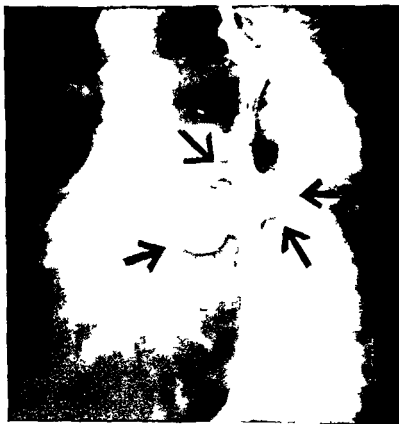


FIG. 57 Multiple diverticula of the esophagus

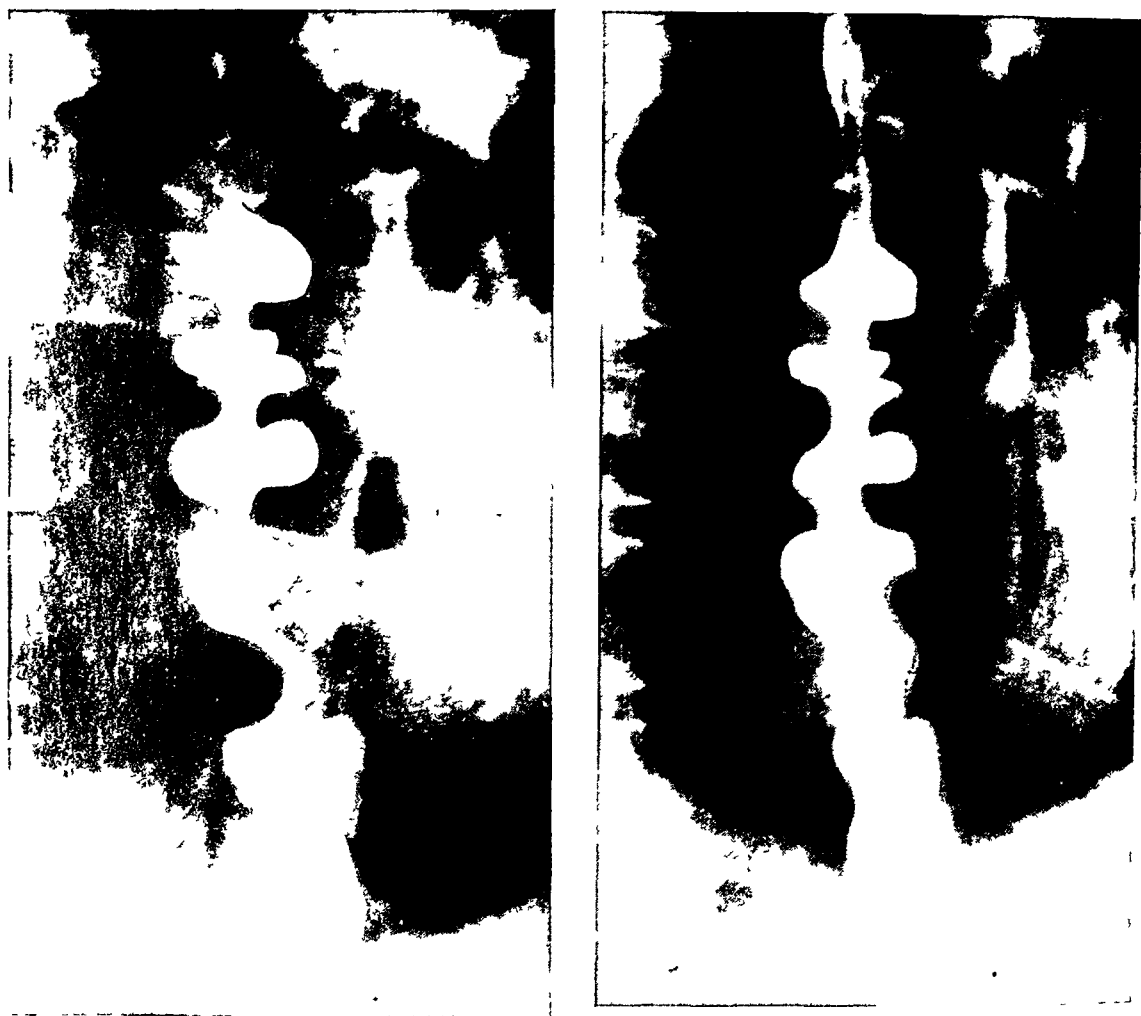


FIG 58 (A, *Left*) Multiple sacculations of the esophagus (B *Right*) Same patient Examination 12 days later Note the tendency to reduplication of the appearance

out its distal half. They involved the anterior and the posterior walls and were apparently circumferential. On re-examination 12 days later, this phenomenon was again noted (Fig 58 B). Note in particular how closely the appearance in Figure 58 B mimics that in Figure 58 A. Therefore, we are dealing with a tendency to reduplication in the formation of these diverticula. This behavior was strongly suggestive of localized factors at the areas where these phenomena recurred. It was difficult to understand how adhesions could produce these findings unless we presupposed that they involved almost the entire length of the esophagus and completely surrounded it, thereby exerting almost simultaneous traction on the wall in every direction.

Mere irritability of the esophagus does

not offer a satisfactory explanation of the tendency to reduplication of the identical pattern. Perhaps the explanation that best accounts for this behavior is the one that assumes that there are areas of diminished resistance in the structural integrity of the wall of the esophagus, perhaps of congenital origin. Then these concentric bulgings would tend to recur time and time again in these regions of diminished resistance under identical conditions of tone and peristalsis.

The superficial undulations in the contour of the esophagus, which behave like ripples, their pattern changing momentarily, may be an entirely different phe-

nomenon from this type of diverticulosis. These superficial ripples may be the expression of irritability or of a disordered form of peristalsis.

A peculiar type of anomalous behavior of the esophagus may be noted in Figure 59. The distal portion is twisted in corkscrew fashion.

REFERENCES

- 1 Ludlow Abr. A Case of Obstructed Deglutition from a preternatural Dilatation of and Bag formed in the Pharynx, in a Letter from Mr Ludlow, Surgeon at Bristol to Dr William Hunter Read August 27, 1764. *Medical Observations & Inquiries* 3 85, 1769.
- 2 Bell Charles. *Surgical Observations*. London, Longman, Hurst, Rees, Orme and Brown, 1816.
- 3 Coffin, M. *Bull Soc anat*, 22 275, 1847.
- 4 Ogle J W. *Tr Path Soc London* 17 141, 1866.
- 5 Meckel J F. *Manual of General Descriptive and Pathological Anatomy*, English translation by Sidney Doane, Philadelphia, Carey and Lea, 1832.
- 6 Zenker F A and von Ziemssen H. *Krankheiten des Oesophagus in Handbuch der speziellen Pathologie und Therapie* (Ziemssen) vol 7, p 1 (Anhang), Leipzig, F C W Vogel, 1877.
- 7 Brintnall E S and Kridelbaugh W W. Congenital diverticulum of the posterior hypopharynx simulating atresia of the esophagus. *Ann Surg* 131 564 1950.
- 8 Gray E D. The radiological demonstration of the potential pharyngeal diverticulum. *Brit J Radiol* 5 640 1932.
- 9 Holmgren Bengt S. Inkonstante Hypopharynxdivertikel. Eine röntgenologische Untersuchung. Supplement to *Acta radiol* LXI 1946.
- 10 Rokitsansky C. *Handbuch der speziellen pathologischen Anatomie*, vol 3 p 160. Vienna 1842.
- 11 Reitzenstein A. Zur Kenntnis und Diagnose der tiefen Oesophagusdivertikel. *München med Wchnschr* 45 354 1898.
- 12 Buckstein J. Multiple sacculations of the esophagus. *J A M A* 86 1128 1926.
- 13 Kjellberg Sven Roland. Retained food remnants in esophageal diverticula resembling tumour and dysphagia in cases of small esophageal diverticula at the bifurcation level. *Acta radiol* 30 436, 1948.
- 14 Hawes J B. 2d. Broncho esophageal fistula and traction diverticulum. *Am J M Sc* 161 791, 1921.
- 15 Westergren A and Ragnell A. Zwei Fälle mit fistulösen Verbindungen zwischen Oesophagus und Atmungsorganen ausgehend von eingeschmolzenen mediastinalen Lymphadenitiden. *Acta med scandinav* 62 519 1925.
- 16 Sigora B. Oesophagusperforation in Verbindung mit Traktionsdivertikel. *Röntgenpraxis* 2 140 1930.
- 17 George G. Oesophageo bronchiale Fistel. *Röntgenpraxis* 4 590 1932.
- 18 Bayer L. Perforation eines Oesophagusdivertikels in der Trachea. *Röntgenpraxis* 6 31, 1934.
- 19 Barsony T and Polgar F. Beiträge zur Röntgensymptomatologie der Hiatusbrüche. *Fortschr Geb Röntgenstrahlen* 37 174 1928.
- 20 Knight G C. The relation of the ex-



FIG 59 Corkscrew esophagus

- trinsic nerves to the functional activity of the oesophagus, Brit J. Surg 22 155, 1934.
- 21 Simon, J Diverticulosis oesophagi und hernia diaphrag hiatus oesophagi, Acta radiol 9 296, 1928
- 22 Havlicek, H Die Grenzdivertikel des Oesophagus und ihre Stellung im biologischen System, Zentralbl Chir 51 2350, 1924
- 23 Fleischner, F Die Divertikel der Speiserohre, Haft oder Adhasionsdivertikel, Fortschr Geb Rontgenstrahlen 45 627, 1932
- 24 Guthrie, K Congenital malformations of the esophagus, J Path & Bact 57 363, 1945
- 25 O'Bannon, R P Congenital partial atresia of the esophagus, Radiology 47 471, 1946

Cardiospasm

Among the earliest reports of cases of dilatation of the esophagus were those by Purton,¹ Hinnay and Rokitsky.

That cardiospasm was due to a failure of relaxation of the cardia rather than to an actual spasm in this region was emphasized by Einhorn³ and Rolleston.⁴ Rolleston's case is of value in that the findings were checked by autopsy. A boy, 8 years old, after an attack of whooping cough, developed intractable vomiting from which he died. The autopsy revealed a markedly dilated esophagus which when distended had a circumference measuring 3 1/2 inches. At the postmortem examination fluid food was still present in the esophagus. The esophagus was 9 1/2 inches long and ran through the thorax in a tortuous manner. The muscular coat was hypertrophied. The mucosa was normal. The lower end of the esophagus was spindle shaped. A finger could be passed from the stomach through the esophageal opening. No evidence of stricture was present either grossly or on microscopic study. The condition was therefore one of primary dilatation. Because of the muscular hypertrophy, Rolleston believed that a functional obstruction might have been present at the cardiac end during life due either to inhibition of the contraction of the longitudinal muscle fibers or to spasm of the circular muscle fibers. Rolleston furthermore brought up the following interesting point. Assuming that the obstruction was spasmodic there should be evidence of hypertrophy of the muscular fibers forming the cardiac sphincter. However, no such evidence was present in this case. He suggested a failure in the coordinating mechanism of the act of swallowing during

which the cardiac sphincter is ordinarily relaxed, as explaining the dilatation and hypertrophy of the esophagus. Paralysis of the longitudinal muscle fibers of the esophagus would lead to dilatation and by interfering with the relaxation of the cardiac sphincter would eventually lead to hypertrophy of the circular muscular fibers.

This condition of failure of relaxation of the cardia has been described by Hurst⁵ as achalasia. According to him the conception of "achalasia of the esophagus" was first broached in the medical literature by Thomas Willis in 1672.

In ten specimens of achalasia of the esophagus Rake (quoted by Hurst) found degenerative changes involving the ganglion cells of Auerbach's plexus. Achalasia of the esophagus thus appears to be due to organic disease of Auerbach's plexus. Another point in favor of the conception of "achalasia" as opposed to cardiospasm is the fact that the cardiac sphincter has never been found hypertrophied at autopsy. Also, in those cases in which operation has been performed, the sphincter within the abdomen is found to be either of normal thickness or actually atrophied, even though the condition has been of long standing.

This work was confirmed by Lendrum,⁶ who made a detailed gross and microscopic study of 13 cases of cardiospasm. In all cases he found that the dilated portion of the esophagus was separated from the stomach by a segment which showed no dilatation, a region which he called "the neck," varying in length from 1.5 to 4.5 cm. There was no evidence of organic narrowing or scarring, of a thickened sphincter, hypertrophic oblique muscle of the stomach,

kinking of the terminal esophagus or of compression of the esophagus by spasm of the diaphragm. In 10 cases the vagus nerve was normal. But in all cases he found a marked loss or complete absence of ganglion cells from the myenteric plexus. The disorder is therefore primarily a disease of the myenteric plexus resulting in failure of relaxation of the cardiac end of the esophagus during the normal act of swallowing.

Von Rosenheim and Holzknecht many years ago described a type of dysphagia due to an intrinsic atony of the esophagus not associated with actual obstruction, with barium sticking to the entire length of the mucosa.

ROENTGEN CHARACTERISTICS

Roentgenologically in cardiospasm, the esophagus shows various degrees of dilatation. It may become enormous in size, and in routine examination of the chest the appearance may simulate that of a heart that has become tremendously enlarged. Infoldings of the esophageal contour may be noted. Fluoroscopically, the esophagus may exhibit exaggerated peristaltic activity in an effort to overcome the failure of relaxation of the cardia.

Differential Diagnosis of the Esophagus in Cardiospasm. The contour is smooth and tapering, ending in the cone-shaped extremity at the cardiac end. This sharpness of outline aids in differentiating the condition from possible malignant infiltration.

Obviously, one must attempt to make certain that the esophagus is empty at the time of examination since food particles retained at the cardiac end may produce translucent defects suggesting organic disease.

Enormous dilatation of the esophagus speaks invariably for a benign cause. When the obstructive lesion is malignant, the esophagus, though it exhibits dilatation

above the area of involvement, as a rule never does so to a very marked degree. This is due to two factors. In the first place a patient with a malignancy of the esophagus dies long before there has been much opportunity for unusual dilatation, whereas, in cardiospasm, the obstruction may continue for many years. In the second place, the obstruction produced by a new growth is as a rule not as marked as in cardiospasm. There is usually a sufficient canalization so that fluids may pass through even though solid foods cannot. Moreover, the sloughing which a malignant tumor frequently undergoes, increases the canalization and consequently diminishes the degree of obstruction, so that unusual dilatation is not quite so apt to occur.

Another aid in differential diagnosis is that while in cardiospasm it is the cardiac end which becomes narrowed, malignant invasion usually affects other regions of the esophagus and, less frequently, the extreme distal portion. Even when this region is involved, the process as a rule is not limited to this area but is more diffuse, with evidence of invasion both proximally and distally in the cardiac region of the stomach. Destruction of the mucosa in "relief" pictures may aid considerably in the differentiation of a malignant infiltrative process from the benign lesion of "cardiospasm."

Illustrative Cases. The following is a case of cardiospasm with a characteristic roentgen appearance and esophagosopic corroboration.

J. S., male, aged 30. The esophagoscope was passed without difficulty down to its lower end. The mucous membrane appeared normal. There was no evidence of foreign body or neoplasm. Three days later the esophagoscope was passed into the stomach without difficulty. No abnormality of the mucosa was noted. The cardiospasm was dilated by bougies passed through the constricted area under esophagosopic control.

Roentgen examination (Fig. 60) revealed marked dilatation of the esophagus with a fluid level near its upper portion. The cardiac

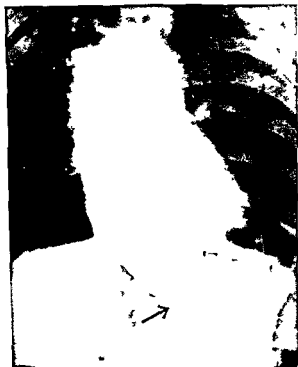


FIG 60 (Left) Cardio pa m with marked dilatation of the esophagus



FIG 61 (Right) Cardio pa sm with tortuous esophagus

end tapered as it entered the stomach and was smooth in outline

From the position of the right border of the dilated esophagus one could readily see

how it might simulate, when not outlined by barium, the right border of an abnormally enlarged heart

The absence of any organic disease asso



FIG 62 (A Left) Cardio pa sm in a child of 10 Examination 6 hours after the administration of the barium suspension Note the abnormal retention of barium within the esophagus (B, Right) Appearance of the child 2 weeks after admission to the hospital

ciated with the obstruction was demonstrated by esophagoscopic observations

Another excellent example of a dilated esophagus due to "cardiospasm" is illustrated in Figure 61. Note the infoldings of the esophagus, which are not uncommonly associated with marked increase in size. Note the smoothly tapering cardiac end of the esophagus with a fine stream of barium making its exit.

"Cardiospasm," while commonly a disease of adult life, may occur in the very young, in rare instances. This is illustrated by the following case:

M. C., female, aged 10. Four months before her admission to the hospital, this child had contracted pertussis. She complained of cough and vomiting. The cough ceased a few days before admission. The vomiting persisted, however, and occurred about once or twice a day, at times immediately after the ingestion of a meal. She did not complain of pain.

Roentgen examination indicated the presence of marked "cardiospasm," with dilatation of the esophagus. Very little of the barium was noted entering the stomach. Within 15 minutes after a hypodermic injection of 1/150 grain of atropine, the esophagus was practically entirely empty of barium.

Three days later, examination of the esophagus 6 hours after the administration of barium (Fig. 62 A) showed retention in the esophagus of half the barium. Thirty minutes after the administration of a hypodermic of 1/150 grain of atropine, the esophagus was again empty of barium.

The esophagoscope was passed to just above the hiatus, which showed considerable thickening of the mucous membrane. At the hiatus there was considerable angulation anteriorly and to the left, with spasm, which made it difficult to pass the esophagoscope into the stomach.

Figure 62 B shows the appearance of the child at the time of admission to the hospital.

The smoothly outlined constriction of the extreme distal end of the esophagus in cardiospasm is clearly shown in the following well-controlled case (M. S., female, aged 24). Eighteen months before admission to the hospital she noticed the onset of gaseous eructations and a tight sensation in her

chest. She had her first pregnancy 5 months later. Several months after that, she complained of almost constant nausea, a feeling of choking and a sticking substernal and epigastric pain. These symptoms gradually became more severe and were made worse after the ingestion of food, either solid or liquid. Vomiting supervened later and would occur immediately after the ingestion of food, either solid or liquid. The only way the patient was able to maintain even a minimal intake was by ingesting very small amounts of liquids at a time. The patient lost 7 pounds in the preceding 3 months.

The patient was of a "nervous" disposition and cried frequently without apparent provocation. At the time of her pregnancy she entered into a common-law marriage. Shortly after childbirth she was separated from her husband. Physical examination was essentially negative. Esophagoscopic examination revealed the following: The opening at the cardiac end of the esophagus was readily identified. The instrument passed into the stomach with ease. There was no spasm or constriction in this region. The mucosa throughout the esophagus was normal. There was no evidence of inflammation, of erosions or ulcerations. The esophagus proximal to the cardiac end was very dilated.

Diagnosis: Achalasia of the cardia of the esophagus.

The roentgen appearance is to be seen in Figure 63. Note the marked narrowing of the extreme distal end of the esophagus where it joins the pars cardiaca of the stomach. This segment is smooth nor is there any irregularity of contour where it joins the more proximally placed, markedly dilated esophagus. There was considerable delay in the escape of the barium from the esophagus into the stomach.

In the following case of cardiospasm radiographic examination of the esophagus shows not only marked dilatation but because of the abnormal elongation the distal segment is transverse in position and is



FIG 63 Cardiospasm (achalasia)
Note the smooth tapering constriction of the cardiac end of the esophagus as it joins the stomach, as well as the marked secondary dilatation of the esophagus proximal to this area



FIG 64 Cardiospasm Note the marked dilatation and elongation of the esophagus and particularly the horizontal distal segment

angulated upon the proximal portion (Fig. 64). The patient (F. T., male, aged 38) gave a history of dysphagia of 17 years' duration. He complained of sensations of fullness in the region of the suprasternal notch following meals, relieved by regurgitation of the food which he had just eaten. His symptoms were also relieved by drinking a glass of water followed by the regurgitation of some of the food. In spite of this he was able to maintain his weight by continuing to eat large amounts of food throughout the day to make up for that which he had vomited. During the preceding 3 months, however, there had been some weight loss. There was no blood in the vomitus at any time. There were no pulmonary episodes due to aspiration of regurgitated food. Prior to the onset of his complaints during a period of from 6 to 12 months he had imbibed raw alcohol while working in a still.

Physical examination was essentially negative. Because of the progressive character of his symptoms an esophagogastricotomy was done. At operation the diaphragmatic hiatus was found to be thickened. The cardia of the stomach and the lower end of the esophagus were hypertrophied, so that passing the finger from above down through the cardia was much like passing it through a tight rectal sphincter. The esophagus above was hypertrophied and dilated and tortuous with a wide bend lying in the right chest. There was a hypertrophic gastritis. There were no ulcerations in the esophagus, and its lumen seemed to be well cleaned out. The distal portion of the esophagus was resected, followed by an esophagogastrostomy. Macroscopic examination of the resected specimen: "The esophagus is dilated, measures 4.5 cm in diameter and the wall is thickened. The cardia measures 2 cm in diameter. The mucosa of the esophagus and the stomach appears normal. Microscopic examination: 'The mucous membrane of the esophagus

and stomach appears normal. The only noteworthy changes are the hypertrophy of smooth muscle, and the numerous clumps of lymphocytes present in the increased and hyalinized fibrous tissue of the lamina

propria and submucosa in the esophagus."

The patient was discharged from the hospital 12 days after the operation in good condition, "eating as he had never done before."

REFERENCES

- 1 Purton, T. An extraordinary case of distention of the esophagus, forming a sac, extending from two inches below the pharynx to the cardiac orifice of the stomach, *London Med & Phys J* 46 540, 1821.
- 2 Hannay, A. J. An extraordinary dilatation (with hypertrophy) of all the thoracic portion of the esophagus causing dysphagia, *Edinburgh Med & Surg* 40 65, 1833.
- 3 Einhorn, M. A case of dysphagia with dilatation of the esophagus, *M Rec* 34 751, 1888.
- 4 Rolleston, H. D. Simple dilatation of the esophagus, *Tr Path Soc London* 47 37, 1896.
- 5 Hurst, A. F. Some disorders of the esophagus, *J A M A* 102 582, 1934.
- 6 Lendrum, F. C. Features of the cardiac orifice of stomach, with special reference to cardiospasm, *Arch Int Med* 59 474, 1937.

Benign Tumors of the Esophagus

Early descriptions of the pathologic appearance of benign tumors of the esophagus have been published by Meckel, Zenker and von Ziemssen, and Cruveilhier

An interesting example of a polyp of the esophagus recorded in the museum of Edinburgh was described by Monro in 1811¹

James Davidson aged 68 was admitted into the Royal Infirmary April 9 1763, for the cure of a polypus in his throat

Upon examining his throat, there was nothing preternatural perceived but, on giving him a vomit, or irritating the fauces, so as to make him retch a large fleshy excrescence was thrown up into his mouth as far as to his fore teeth consisting of four different fangs joined together by one common root These were of a pretty firm fleshy texture possessed of a good degree of elasticity He could hardly allow them to remain half a minute in his mouth as they shut up the larynx and thereby entirely stopt his breathing

This polypus had for several years prevented him from swallowing any thing without much difficulty neither could he breathe so freely nor speak so distinctly as usual it like wise occasioned a cough which frequently forced the polypus into his mouth

A part of the polyp was removed by a snare but he died two years later

On dissecting his body the oesophagus was found to be greatly dilated by a very large fleshy excrescence or polypus which grew out from its fore part by a single root about three inches lower than the glottis but was split at its under part into several lobes the largest and longest of which reached down to the upper orifice of the stomach

A polypoid myoma of the esophagus was described in 1872 by Coats in a male aged 61 who complained of increasing dysphagia

and substernal pain upon swallowing Autopsy revealed a large polypoid myoma lying within the lumen of the esophagus and attached to the posterior wall by a pedicle 1¾ in long

The first specimen of a myoma of the esophagus to be presented before the London Pathological Society was by Fagge in 1874² The specimen had been obtained from the body of a man aged 38, without any record of dysphagia in the clinical history

Autopsy disclosed a large egg shaped tumor within the layers of the esophagus growing from the anterior wall just below the level of the bifurcation of the trachea The surface of the tumor which lay beneath the mucous membrane was smooth and rounded The membrane over it was freely movable and could be dissected away from it very easily The other surface which lay within the longitudinal muscular coat was slightly nodulated The muscular fibers could be followed over the tumor but part of it was inseparably adherent to them On cut section the tumor resembled a myoma of the uterus The diagnosis was confirmed by microscopic examination The specimen is preserved in the Museum of Guy's Hospital

The first case of a rhabdomyoma of the esophagus was described by Wolfensberger in 1894³ The lesion was discovered at autopsy in a 75 year old emaciated male who had complained of dysphagia but was able to swallow liquids The tumor was polypoid, originated in the distal portion of the esophagus and extended into the stomach

The rarity of benign tumors is shown in the Bellevue Hospital records during the

period 1905 through 1935, which disclosed 16 cases of benign tumor of the esophagus. Of these, 9 were found at autopsy out of a series of 22,810 autopsies conducted during this period. These tumors were classified as follows: papillomas, 4; fibromas, 3; myomas, 2; lymphangiomas, 2; polyp, 1; leiomyoma, 1; and angioma, 1. Two of the tumors found at autopsy were not classified.

Patterson⁷ stated that in a review of the literature from 1712 to 1932 she found 61 cases of benign tumors of the esophagus, to which she added a case of a myxofibroma. Only 42 were reported in any detail and even in some of these cases description was incomplete. (The article contains no bibliography of references to these cases.) Although the lesion is quite rare, there has been a considerable increase in the number of cases recorded since 1933, and in the report by Adams and Hoover in 1945⁶ 35 additional cases were recorded in the literature (including 3 of their own), thus making a total of 97 cases. To these may be added the 16 cases referred to above, found at Bellevue Hospital.

ROENTGEN DIAGNOSIS

The essential findings in the demonstration of an intraluminal benign tumor of the esophagus are the following:

1. The tumor usually produces a rounded, centrally located translucent area corresponding to the intraluminal position of the growth. This is due to the anatomic characteristics of benign tumors generally, which hold for tumors of the esophagus as well. This rounded, translucent appearance produced by a benign tumor is shown in the cases reported by Pape and Spitznagel,⁷ Beutel,⁸ Palugyay,⁹ and others.¹⁰⁻¹³ Exceptionally, the tumor may be of such nature that the roentgen appearance may simulate that of a proliferating malignant tumor. This occurred in Zehbe's¹⁴ case, in which roentgen examination of the esophagus disclosed a thin layer of barium passing along the lateral walls and encompassing several

dense areas of different form and size. At autopsy, however, a large polyp was found, the size of a cucumber, which hung by a pedicle from its anterior wall and filled almost the entire esophagus. Histologic examination showed it to be a fibroma.

The diagnosis is further complicated by the fact that sarcomas of various types may appear in some cases as rounded, well-circumscribed, intraluminal tumors, sometimes pedunculated. Thus the sarcomas reported by Ogle,¹⁵ Albrecht,¹⁶ Stephan,¹⁷ von Hacker¹⁸ and Dvorak¹⁹ were pedunculated or polypoid.

In Sommers' case,²⁰ roentgen examination showed a translucent area within the lumen of the esophagus, outlined by barium. Autopsy showed a sausage-shaped tumor 14 cm. long within the lumen of the esophagus fixed to the wall by a short pedicle. Histologic study, however, showed the tumor to be a carcinosarcoma.

Similarly, Krieglstein,²¹ described the roentgen findings of an intraluminal elliptical area within the esophagus, the size of a goose egg, outlined by barium, which at autopsy proved to be due to a fibroma with sarcomatous changes.

Also, in the carcinosarcoma reported by Stout, Humphreys and Rottenberg,²² radiographic examination showed a polypoid type of intraluminal deformity of the distal portion. The resected tumor was pedunculated with only a superficial attachment to the anterior wall of the lower portion of the esophagus. Histologic study showed evidence of both carcinomatous and sarcomatous elements. The authors in a review of 10 other neoplasms of a similar nature reported in the literature, noted that they were all polypoid tumors. They do not include the case of carcinosarcoma reported by Sommers²⁰ which, as already stated, was also a polypoid tumor.

The fibrosarcoma reported by Clark²³ was a polypoid tumor attached by a pedicle to the posterior wall of the proximal portion of the esophagus. It produced an intraluminal area of translucency indistinguish-

able from any other type of polypoid tumor. The diagnosis was established by histologic study of the resected lesion.

Similarly, the two sarcomas of the esophagus reported by Lortat Jacob and Badaro⁴ were intraluminal and attached by a pedicle. The mucosa covering the tumor was essentially intact but showed secondary ulcerations. The roentgen appearance simulated those features produced by a benign intraluminal polyp.

1 While exceptionally, therefore, the differential diagnosis between a benign tumor and a malignant lesion may offer serious difficulty, the well circumscribed character of the translucent area within the lumen of the esophagus speaks definitely in favor of the benign nature of the lesion. As the barium passing down the esophagus reaches the tumor, there is a splitting of the stream, a thin layer of barium passing between the tumor and the walls of the esophagus, thereby encompassing the mass. In the event that the tumor is small, a thick suspension of barium may be objectionable in that it may actually obscure the lesion. At times, therefore, a thin barium water suspension may aid in the visualization of such a lesion.

2 When the tumor is intraluminal in position, the radiologic appearance of the esophageal contour remains smooth and exhibits no evidence of irregularity. An important point is the fact that the mucosal folds of the esophagus are intact. Such evidence is of considerable value in differential diagnosis between a benign lesion and a malignant infiltration.

3 If the tumor has a long pedicle, there may be variation in the exact position of the tumor at different times and with change in the position of the patient. More over in rare cases the pedicle of the tumor itself may be visualized as a translucent zone through the contrast medium in the esophagus. This was well demonstrated in the carefully controlled case reported by Tamiya and Nosaki.

Because of the marked degree of mobility

possessed by some of these benign tumors of the esophagus, they may actually be brought up into the mouth as illustrated in the case reported by Samson and Zelman²⁶ and in one case described by Weyrich, a pedunculated lipoma of the esophagus choked a patient to death by closing off the opening to the larynx.

4 The tumor may produce considerable dilatation of the esophagus when present at or near the cardiac end. This occurred in the case reported by Haensch²⁸. A mobile tumor attached by a pedicle, by invaginating the cardiac end of the esophagus, may be a cause of temporary obstruction at the cardiac end with secondary dilatation. In a case of this nature, roentgen examination at one time may exhibit a typical rounded translucent area within an esophagus the lumen of which is normal, and reexamination at a later time may show marked obstruction of the cardia. Owing to this dilatation of the esophagus as a result of obstruction and the failure of the barium to make its exit into the stomach the barium suspension may fill up the dilated esophagus thereby obscuring the tumor and arousing the suspicion of cardiospasm as a cause of the condition.

5 The determination of the point of origin of a benign tumor of the esophagus, as to whether it is mucosal or submucosal is extremely difficult and in many cases impossible. One of the reasons is that pathologically, a tumor originating within the wall of the esophagus may grow indirectly into the lumen and be indistinguishable from an intraluminal tumor of mucosal origin certainly as far as the roentgen appearance is concerned. When the tumor shows a rounded mass extending into the mediastinum away from the wall at the same time that it projects intraluminally, one may entertain a strong suspicion that the mass is an intrinsic lesion of extra-mucosal origin. The fact that this mass moves with the changes in the position of the esophagus adds further support to this diagnosis. However, extra esophageal

masses which have become closely adherent to or have actually infiltrated into the wall of the esophagus may make differentiation from a primary lesion of the esophagus extremely difficult and often impossible

The serious technical difficulties involved are well demonstrated by the experimental efforts as well as the clinical observations of Schatzki and Hawes²⁹

A classical deformity of the esophagus produced by the intramural submucosal leiomyoma is to be found in a number of reports Calmenson and Clagett,³⁰ Hurwitz,³¹ Goldman and Masters,³² Bigger, Kay and Vinson³³ In the case reported by Calmenson and Clagett,³⁰ in addition to a smooth sharply defined defect of the anterior wall of the esophagus produced by the larger leiomyoma there was a small rounded intraluminal translucent area produced by a second, smaller leiomyoma Depending on the exact anatomic location of the tumor in relation to the esophagus, it may appear as an intraluminal lesion in-

distinguishable from a tumor of mucosal origin^{33 34}

Differential Diagnosis In spite of all the difficulties that may arise, the differentiation of a benign tumor from a malignant new growth depends primarily on the well-circumscribed, intraluminal translucent area produced by the lesion, with a smooth contour of the wall of the esophagus and an intact mucosa Mobility of this area also favors the diagnosis of a benign tumor attached by a pedicle

In malignancy of the esophagus, as will be discussed more fully later, the contour of the wall as a rule is irregular and rigid, the mucosal folds are destroyed, and a proliferating mass, if present, extending into the lumen, produces translucent areas, frequently of a finger-printing type and irregular in outline Moreover, a mass of this nature is fixed and is not apt to exhibit evidence of change of position within the esophagus

Translucent areas within the esophagus



FIG 65 (A, *Left*) Benign tumor of the esophagus Note the well-rounded translucent area produced by the tumor within the confines of the distal end of the esophagus (B, *Right*) Same patient The tumor is obscured by a large amount of barium Note also the dilatation of the esophagus, apparently produced by occlusion of the lumen by the tumor

may be caused by swallowed air trapped within the column of barium. The differential diagnosis, however, should offer no difficulty, as translucent zones produced in this manner are of an inconstant character and may not be demonstrated on re-examination. Under fluoroscopic control the

swallowed air may be noted passing through the course of the esophagus into the stomach.

Illustrative Case The following is an example of a benign tumor of the esophagus.

S F, female aged 50. For about 8 years this patient had been complaining of attacks of substernal pain radiating to the back. These attacks might occur at any time, without any relation to meals. At times, the pain was extremely severe. On one occasion, the pain lasted throughout an entire night, finally becoming so severe that she thought she was dying. She vomited at times. There was never a history of dysphagia. Her weight fluctuated from time to time, weight loss being only temporary and followed by gain in weight.

Roentgen examination of the esophagus (Fig. 65 A) revealed a rounded translucent shadow at the cardiac end, having the characteristics of a benign tumor. Note also that Figure 65 B shows the esophagus to be moderately dilated. Owing to the large amount of barium present, the tumor is obscured.

FIG. 66 (A, Top) Leiomyoma of the esophagus. Note the fairly sharply defined concave defect of the anterior wall and the soft tissue shadow extending beyond the confines of the esophagus produced by the tumor.

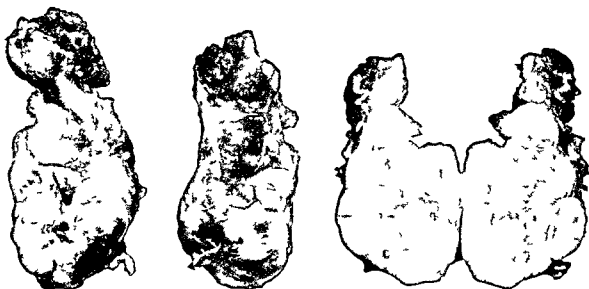


FIG. 66 (B Left; C Center and D Right) Same patient as is shown in Figure 65 A. Different views of the tumor removed at operation.

Repeated esophagoscopic examinations revealed a polyp 36 cm from the upper incisor teeth and originating from the anterior wall of the esophagus. A biopsy was done. Histologic examination revealed no evidence of malignancy.

The dilatation of the esophagus seen in Figure 65 B may best be explained by a temporary mechanical blockage of the cardiac opening by the polyp. This may also explain the clinical symptoms of recurrent attacks of severe substernal pain and vomiting.

A benign intramural extra-mucosal tumor of the esophagus is illustrated by the following fully controlled case.

J S, male, aged 54 (patient of Dr. Tolks). The patient gave a 3 months' history of constant epigastric pain, worse, however, shortly after meals. It was relieved by belching and the application of heat. In addition, he complained of a feeling in the midchest as if swallowed food could not go down. He had lost 12 pounds.

Physical examination was essentially negative.

Roentgen examination of the esophagus (Fig. 66 A) revealed the following features:

1. A fairly smooth, localized, concave defect involving the midportion of the anterior wall.

2. This area was observed to show slight

changes in its contour in various films. The posterior wall of this segment of the esophagus was entirely normal, and peristalsis traversed this region.

3. Of great importance was the fact that an elliptically shaped, soft-tissue shadow occupied the area of the concave defect.

4. Particularly significant was the fact that this elliptical shadow extended beyond the confines of the lumen of the esophagus itself.

Based on these findings the diagnosis was made of a benign, submucosal tumor displacing the mucosa toward the opposite wall of the esophagus and at the same time extending beyond the confines of the esophagus itself. It was also stated that this tumor would prove to be a leiomyoma of the midportion of the esophagus.

The patient was operated on by Dr. Nissen. Through the left thoracic cavity the esophagus was exposed, revealing a large, hard tumor occupying its midportion approximately between the level of the bifurcation and the inferior border of the aortic arch. The muscular layer of the esophagus overlying the tumor was incised. It then became evident that the tumor did not infiltrate the muscular layer, the submucosa or the mucosa. The tumor was dissected out, leaving the mucous membrane intact. The appearance of the tumor is shown in Figures 66 B, C and D.

Histologic examination of the tumor corroborated the preoperative diagnosis of leiomyoma.

REFERENCES

1. Monro, Alexander. *The Morbid Anatomy of the Human Gullet, Stomach and Intestines*, p. 186, Edinburgh, Constable, 1811.
2. Coats, Joseph. Large polypoid myoma of the esophagus, *Glasgow M J* 4 201, 1872.
3. Fagge, C. Hilton. Case of myoma of the esophagus, *Tr Path Soc London* 26 94, 1875.
4. Wolfensberger, R. Ueber ein Rhabdomyom der Speiseröhre, *Beitr path Anat* 15 490, 1894.
5. Patterson, Ellen J. Benign neoplasms of the esophagus, *Pennsylvania M J* 36 244, 1933.
6. Adams, Ralph and Hoover, W. B. Benign tumors of the esophagus, *J Thoracic Surg* 14 279, 1945.
7. Pape, R., and Spitznagel, K. Über Ösophagusmyome, *Fortschr Geb Röntgenstrahlen* 44 616, 1931.
8. Beutel, A. Benigner, wahrscheinlich zystischer Tumor des Ösophagus, *Röntgenpraxis* 4 814, 1932.
9. Palugyay, J. Röntgendiagnose gestielter Speiseröhrengeschwülste, *Röntgenpraxis* 4 761, 1932.
10. Beeler, R. C., Collins, J. N., and Hall, M. F. Benign pedunculated tumors of the esophagus, *Am J Roentgenol* 60 466, 1948.
11. Vinson, P. P., Moore, A. B., and Bowling, H. H. Hemangioma of the esophagus. Report of a case. *Am J M Sc* 172 416, 1926.
12. Engelking, C. F., Knight, M. D., Brauns, W. H., and Herschberger, L. R. Benign

- tumors of the esophagus report of a case of neurofibroma Arch Otolaryng 52 150, 1950
- 13 Schinz, H R, Baensch, W E, Iriedl, E, Uehlinger, E Lehrbuch der Rontgen diagnostik, p 3027, Stuttgart, Thieme, 1952
 - 14 Zehbe, M Oesophagusstenose durch gutartigen tumor (polyposis), Fortschr Geb Rontgenstrahlen 32 430 1924
 - 15 Ogle, C Sarcoma of esophagus, Tr Path Soc London 47 40, 1896
 - 16 Albrecht, H Polyp des Oesophagus, Wien med Presse 36 666, 1895
 - 17 Stephan B H Zur Casuistik der Dysphagie bei Kindern (sarcoma oesophagi bei einem 4 jährigen Knaben), Jahrb Kinderh 39 354, 1890
 - 18 von Hacker V Zur Kenntnis des Oesophagussarkoms, Mitt a d Grenzgeb d Med u Chir 19 396, 1908
 - 19 Dvorak, H J Sarcoma of the esophagus Arch Surg 22 794, 1931
 - 20 Sommers J Ein Beitrag zur Diagnostik der Speiseröhrentumoren Fortschr Geb Rontgenstrahlen 31 26 1923
 - 21 Kneeglein, Fr Eine gestielter polyposen Tumor des Oesophagus Frankfurt, Ztschr f Pathologie 50 1, 1936
 - 22 Stout A P Humphreys G H II and Rottenberg L A A case of carcino sarcoma of the esophagus Am J Roentgenol 61 461 1949
 - 23 Clark D E Sarcoma of the esophagus report of successful resection of a fibro sarcoma Arch Surg 59 148 1949
 - 24 Lortat Jacob J L and Badaro Deux sarcomes primitifs de l'oesophage p.d. cules Semaine Hop Paris 26 1743, 1950
 - 25 Tamiya C, and Nosaki S Diagnose und Therapie gestielter Oesophagustumoren Fortschr Geb Rontgenstrahlen 49 481, 1934
 - 26 Samson, P C, and Zelman, J Pedunculated tumors of the esophagus, Arch Otol 36 203, 1942
 - 27 Wevrich, Gunther Plotzlicher Tod durch ein gestieltes Lipom der Speiseröhre, Deutsche Ztschr ges gerichtl Med 21 164 1933
 - 28 Haenisch F Beitrag zur Rontgendiagnostik des Oesophagus benigner Oesophagustumor Fortschr Geb Rontgenstrahlen 32 432, 1924
 - 29 Schatzki, R, and Hawes, L E The roentgenological appearance of extra mucosal tumors of the esophagus Am J Roentgenol 48 1, 1942
 - 30 Calmenson, M and Clagett O T Surgical removal of leiomyomas of the esophagus Am J Surg 72 745, 1946
 - 31 Hurwitz Alfred Leiomyoma of the esophagus Report of a case Surgery 25 304, 1949
 - 32 Goldman A, and Masters H Leiomyoma of the esophagus Arch Surg 60 559, 1950
 - 33 Bigger I A, Kay, W R and Vinson P P Leiomyoma of esophagus Report of case Virginia M Month 77 290 1950
 - 34 Case Records of the Massachusetts General Hospital Case 33252, New England J Med 236 957 1947

Malignant Lesions of the Esophagus

Carcinoma of the esophagus may be classified mainly according to two types. The first is the scirrhus variety. In its earliest stages this is submucosal in location, causing a protrusion of the mucosa into the lumen. Its gradual encroachment finally produces an annular stenosis. At a later stage there may be invasion of the lumen and ulceration of the mucosa.

In the second type, there is an encroachment on the lumen of the esophagus by an expansive type of growth with an irregular cauliflowerlike contour and secondary ulceration. Stenosis is produced by occlusion of the lumen. Peristalsis is absent, the wall becomes rigid, and the lumen is narrowed. There is a greater tendency in this type of malignant lesion for destructive changes to occur. As the result of the breaking down of the tumor, there may be an increase in the lumen of the esophagus and a temporary alleviation of symptoms due to obstruction. Malignancy of the esophagus remains localized for a long time.

The carcinoma may also invade any of the neighboring structures, either through extension of the growth or because of perforation. The carcinoma may invade the wall of the trachea or the bronchi. It may reach the cardiac area, the pericardium, the pleura and the lungs. The adventitia of the aorta may be infiltrated. The vertebrae may be invaded and destroyed. Tumefaction entering the vertebral canal may compress the spinal cord and lead to fatal paraplegia.

Metastatic spread by way of the lymphatics and blood vessels may involve the lymph nodes, the liver, the lungs, the kidneys, the adrenals, the pancreas and the bones.

ROENTGEN DIAGNOSIS

A knowledge of the underlying pathology leads to a clearer understanding of the roentgen deformity of the esophagus produced by malignant lesions in this region. When the infiltrative process is limited to the wall of the esophagus, the contour is irregular, the wall appears stiff, peristalsis is absent, and the lumen is narrowed. The mucosa shows abnormalities as a result of the submucosal infiltration and secondary ulceration. Proximal to the obstruction, the esophagus becomes dilated. In the proliferative type of malignant new growth, the lumen becomes directly invaded, the mucosa is destroyed, and irregularly outlined translucent areas are present within it. Almost invariably there is an associated irregularity in the contour of the involved region, and the evidence of adenomatous invasion is associated with destructive changes produced by infiltration of the wall itself. In such cases, also, the lesion leads to a narrowing of the lumen and dilatation of the esophagus proximal to the site of involvement.

The dilatation of the esophagus, however, is never very great and never reaches the degree seen in those cases of obstruction produced by a lesion that is benign. Two reasons explain this fact. In the first place, a patient with malignancy of the esophagus does not survive sufficiently long for extreme dilatation of the esophagus to take place. The second reason is that, owing to the breaking down of the tumor, the obstruction is usually incomplete. When the obstruction is due to a benign lesion, however, as in cardiospasm, the condition may progress for many years. Moreover, the

obstruction is more nearly complete, and no canalization develops, as in the breaking down of the tissue of a malignant tumor

It is for this reason also that the degree of dilatation of the esophagus and the evidence of obstruction may show considerable diminution at times in the case of a malignant lesion. The sloughing of a large mass may eliminate the cause of obstruction.

While, as a rule, malignant lesions of the esophagus are single, in rare instances two malignant lesions may be present with evidence of normal intervening tissue as determined both roentgenologically and on pathologic examination.

Another important roentgen finding is due to the fact that malignant lesions of the esophagus may perforate neighboring tissue, and particularly a bronchus. Under such conditions an irregularly outlined barium shadow will be seen extending beyond the confines of the growth itself for a variable distance. In those cases in which the bronchus has been perforated by the growth barium will be noted making its exit through the fistulous communication and outlining the tracheobronchial tree. In this connection, it is important to remember that, as a result of the stenosis produced by the new growth, particularly when this is high in location, there may be a regurgitation of barium up into the pharynx and then into the trachea and the bronchi. Unless the patient has been fluoroscoped with considerable care the findings may be misinterpreted as being the result of an actual fistulous communication between the esophagus and the trachea or the bronchus. By observing the behavior of the esophagus as the patient drinks the barium, it may be possible to determine which of the two factors is at fault. When a fistula is present, the actual exit of the barium from the new growth may be noted. Because of the danger that the patient with a stenosing malignant lesion may regurgitate the barium during examination it is important that only a small amount be administered. A teaspoonful of the barium-acacia mixture may be sufficient and, as

previously emphasized, if clinical evidence of stenosis is marked, a thinner barium water mixture, possibly only slightly thickened with acacia, may be employed.

In some cases, it is possible for a tracheo-esophageal fistula to be present and yet go unrecognized roentgenologically. Either the area of communication may be extremely small or it may be so walled off by edema and inflammation as to defy detection.

Differential Diagnosis. The differential diagnosis between a benign and a malignant tumor has been considered previously in the discussion of benign tumors of the esophagus.

Ordinarily the differentiation from cardiospasm should offer no difficulties. Malignant invasion may occur almost anywhere in the course of the esophagus, although the site of predilection is more apt to be at the points of normal constriction. In cardiospasm, the lesion is limited to the cardiac end of the esophagus. Limitation of a malignant infiltration to the cardiac end of the esophagus does occur, but such localization is not common, except as an extension from a carcinoma of the cardiac end of the stomach. In malignancy there is irregularity of contour and destruction of normal mucosal folds. In cardiospasm, the contour is extremely smooth and tapers to a fine line. Food retention may produce irregularity of contour in cardiospasm and this is one reason why the patient being examined should have had no food for at least 12 hours. Re-examination of the patient in such cases, if he is properly prepared, will show that the irregularity in cardiospasm is no longer present.

Moreover, as already stated the degree of dilatation of the esophagus in cardiospasm may be enormous. In new growth this does not become nearly so marked. If the dilatation is extreme one may safely assume that the cause is due to cardiospasm.

Considerably greater difficulty in differential diagnosis may arise in some cases when the stenosis of the esophagus is produced by the action of a corrosive. While

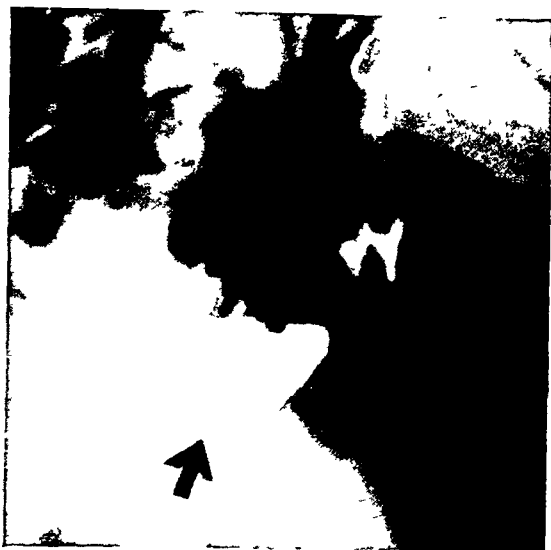


FIG 67 Carcinoma at the pharyngo-esophageal junction

the effect of the corrosive may be such as to produce an irregularity involving almost the entire length of the esophagus and therefore readily recognizable as having been produced in this manner, a localized area of destruction and irregular narrowing may closely simulate the roentgen appearance of a deformity produced by a new growth. In such borderline cases the clinical history will be the decisive factor in differentiating the two conditions.

The appearance of the esophagus produced by other organic lesions of an intrinsic nature will be discussed in later chapters.

Illustrative Cases A carcinoma may be present at the mouth of the esophagus.

S. F., male, aged 66. The patient gave a history of dysphagia of 18 months' duration, ultimately he had difficulty in swallowing even liquids. He had lost considerable weight. He stated that at the onset of his distress a lump had been removed from the left lower cervical region and that examination showed this to be malignant.

Physical examination revealed a pallid, white male, showing evidence of recent weight loss. An operative scar was present in the left lower cervical region. The examination was otherwise essentially negative. Endoscopy revealed the cricopharyngeus region to be friable and hard. A biopsy was taken from the area of apparent tumefaction.



FIG 68 Carcinoma of the esophagus

The pathologic diagnosis was squamous-cell carcinoma of the esophagus.

Roentgen examination (Fig 67) revealed complete obstruction to the flow of barium at about the junction of the hypopharynx and the esophagus, with a cone-shaped defect.

The roentgen diagnosis, then, was that a mass was causing obstruction at the pharyngo-esophageal junction. Pathologic examination, as noted above, showed the tumor to be a squamous-cell carcinoma.

The next case is that of a carcinoma of the proximal portion of the esophagus.

A. W., male, aged 52. The patient had complained of increasing dysphagia for 2 months. Prior to that he had complained of a productive cough, occasional night sweats and weakness.

Roentgen examination of the chest showed chronic pulmonary tuberculosis.

Esophagoscopy showed a new growth 20 cm from the teeth. The growth was irregular, firm, cauliflowerlike in appearance, bleeding readily to the touch. Biopsy showed the lesion to be a squamous-cell carcinoma.

Exploratory thoracotomy revealed the following facts. From the aortic arch upward the periesophageal tissues were invaded, and



FIG. 69 Carcinoma of the esophagus

the esophagus was fixed by inflammatory and probably neoplastic tissue. The hilus of the left lung was also adherent in this area. The condition was regarded as inoperable, and the chest was closed without drainage.

Roentgen examination of the esophagus (Fig. 68) revealed an irregular annular narrowing at the junction of the upper and the middle thirds, with destruction of the normal mucosal markings, classical evidence of a new growth.

When the carcinoma of the esophagus is high in location the pharynx may be distended with barium. This is illustrated by the following case.

L. L., male, aged 67. About 2 months be-



FIG. 70 Carcinoma of the esophagus
Note the 'finger printing' deformity

fore his admission to the hospital this patient complained of the feeling of a lump in his throat on swallowing. He described the lump as being at the level of the cricoid cartilage. He had increasing difficulty in swallowing. Finally 3 weeks before admission, he was unable to swallow even liquids in any but minute amounts. The swallowed food was regurgitated almost at once. He had lost 30 pounds in the preceding 3 weeks.

The patient was markedly emaciated. The left pupil was larger than the right and both were of irregular contour. There was a feeling of resistance, due to a mass deep in the neck.

Esophagography showed a large fungating mass in the proximal portion of the esophagus. The diagnosis on microscopic examination was squamous-cell carcinoma.

Roentgen examination (Fig. 69) revealed evidence of an annular malignant lesion with marked obstruction involving the proximal portion of the esophagus about 1 inch distal to the pharyngo-esophageal junction. As a result of the obstruction the entire pharynx was distended with barium. Note that the esophagus proximal to the area of narrowing is distended in such a way that it simulates the appearance of a diverticulum.



FIG 71 Carcinoma of the cardiac end of the esophagus

The following is an example of carcinoma of the middle third of the esophagus

J M, male, aged 58 This patient gave a 4 months' history of difficulty in swallowing, this gradually increased in severity and eventually became very painful Pain was retrosternal and radiated down to the epigastrium Occasionally he vomited the food just eaten He had lost 40 pounds Esophagoscopy examination revealed the following The esophagoscope was passed to a point 29 cm from the upper margin A fungating soft mass was seen obstructing the lumen A small bougie was passed below this obstruction. A specimen was removed for biopsy The pathologic diagnosis was squamous-cell carcinoma of the esophagus

Roentgen examination (Fig 70) revealed a diffuse narrowing of approximately the middle third of the esophagus, with irregularly outlined translucent areas within this region There was moderate obstruction of the esophagus above the area of involvement The roentgen diagnosis was carcinoma of the esophagus

Less commonly a carcinoma of the esophagus may be limited to the cardiac end, as in the following case

J G, male, aged 63 This patient com-

plained of the sudden onset of cramps in the epigastrium 7 weeks before admission to the hospital At the time of admission, the pain was continuous and was aggravated by eating Vomiting had continued until 2 weeks before He had extreme difficulty in swallowing and could retain only water He lost 15 pounds in 2 months

At operation, a hard, annular tumor surrounding the cardiac portion of the stomach was found It extended downward from the diaphragmatic opening for a distance of about 6 cm along the circumference of the stomach There were many small, nodular white masses in the liver, evidently secondary deposits from this growth

Roentgen examination of the esophagus (Fig 71) revealed a moderate degree of obstruction at its junction with the cardiac end of the stomach There was considerable irregularity of contour at the cardiac end, which is characteristic of new growth

Rarely, multiple malignant lesions of the esophagus may occur The following is an example of this

F. W., male, aged 49 About 6 weeks prior to his admission to the hospital, this patient had begun hiccoughing at about 2 P M, continuing for about 2 hours This had recurred almost every afternoon since the onset It was occasionally relieved by drinking from 2 to 3 glasses of water The patient lost 36 pounds in 2 months Esophagoscopy revealed a definite annular constriction about 12 cm below the cricopharyngeus muscle The area was pale and friable and it bled easily Gastrostomy revealed that the stomach was markedly dilated There were firm, nodular masses extending along the lesser curvature of the stomach to the duodenum

The autopsy revealed the following "Opposite the bifurcation of the trachea on that surface of the esophagus, there is a large ulcerated, indurated, necrotic lesion about 7 cm by 4 cm in size The ulcerative process has extended to, but not into, the trachea and aorta All but a small portion of the circumference of the esophagus is involved in this lesion A similar lesion involves the esophagus at the cardiac orifice of the stomach and appears to have extended down into the fundus of the stomach, at which point it is perforated posteriorly into the lesser peritoneal cavity

"Microscopic examination of the esophageal tumor showed ulceration of the epithelium and invasion of the muscularis, with tumor



FIG 72 (A, *Left*) Carcinoma of the esophagus (B, *Right*) Same patient Note the independent new growth at the cardiac end of the esophagus

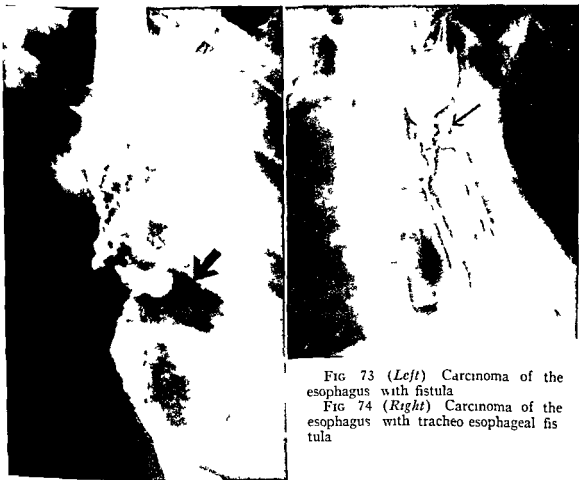


FIG 73 (*Left*) Carcinoma of the esophagus with fistula

FIG 74 (*Right*) Carcinoma of the esophagus with tracheo esophageal fistula

cells singly and in masses. The cells were polyhedral, variable in size and granular. The nuclei were large, oval or circular. Mitotic figures were fairly common.

The diagnosis was "transitional-cell epithelioma."

Roentgen examination revealed an irregularly outlined, markedly narrowed area of the esophagus involving the middle third. There was considerable dilatation of the esophagus above this region (Fig 72 A). The lower third of the esophagus again assumed a normal appearance as far as the cardiac end. In this region there was evidence of neoplastic infiltration. Particularly significant was a translucent area at the extreme cardiac end of the esophagus, encompassed on either side by a thin stream of barium, which was best seen in the examination of the stomach (Fig 72 B), indicating a second tumor.

A serious complication of malignancy of the esophagus is the development of a fistula into the periesophageal tissues (Fig 73). This may ultimately perforate into adjacent structures, particularly into the trachea or bronchus (Fig 74).

A fistulous communication between a malignancy of the esophagus may be present and yet may not always be demonstrable roentgenologically. This is seen in the following example.

About 6 months before his admission to the hospital, this patient, for the first time, had had difficulty in swallowing solid food. Swallowing was accompanied by pain in the chest. Occasionally he had difficulty with liquids as well. He vomited on several occasions, but the

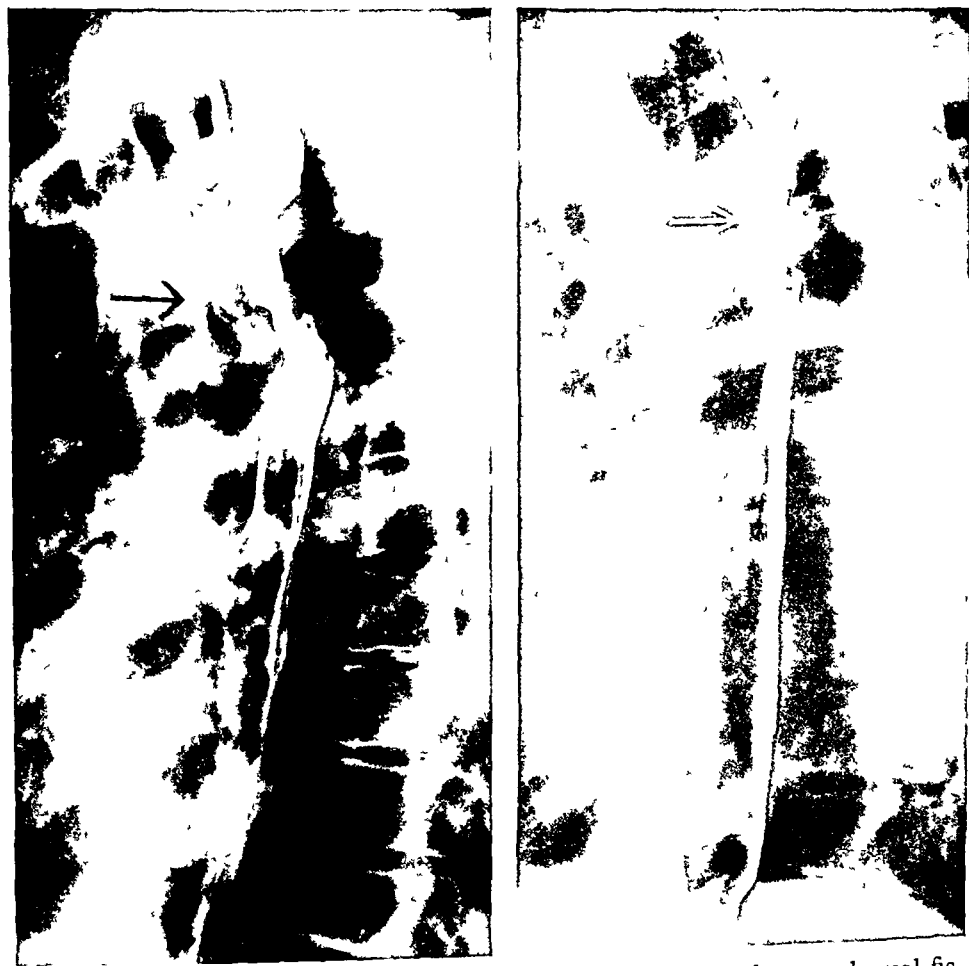


FIG 75 (A, *Left*) Malignancy of the esophagus, with tracheo-esophageal fistula showing at first examination. (B, *Right*) There is no evidence of the tracheo-esophageal fistula at this later examination, although the fistulous communication was found subsequently at autopsy.

vomitus was never bloody. He lost 15 pounds in 6 weeks. His temperature was 101°F .

Esophagoscopic examination revealed a carcinoma involving the bronchi and the esophagus. At the point of the fistula, the lumen was narrowed. A gastrostomy was done.

The autopsy revealed the following: About 7 cm below the epiglottis the mucous membrane of the esophagus is discolored and ulcerated, and the tissues are inelastic and hard. Carcinomatous change is rather extensive, encircles the esophagus completely and extends over an area distally of from 6 to 7 cm. Crypts and pockets have been formed in the esophageal wall irregularly throughout the tumor. The lumen of the esophagus is only moderately constricted over the affected zone. Near the lower portion of the tumor is a perforation having diameters of 1 cm by 0.5 cm, which communicates directly with the trachea about 5 cm above the tracheal bifurcation. At the point of perforation, as well as throughout the tumor zone the trachea is densely fused to the esophageal wall, the adhered area being stone hard and inelastic.

The microscopic examination of the esophagus showed the squamous cell epithelial lining to be missing in many places. Where it is present it is orderly and regular. The subepithelial stroma is greatly thickened and densely infiltrated with lymphocytes, fibroblasts and plasma cells. In several areas the squamous cell lining is replaced by masses of amorphous necrotic tissue, throughout which no malignant tissue can be distinguished. The diagnosis is chronic inflammatory tissue with necrosis (postradiation). Cervical lymph nodes. The lymphoid tissue is generally sparse throughout and the fibrous tissue prominent. Several zones are seen where the lymph structures are completely replaced by nests of invading epithelial cells which appear definitely squamous in character. This indicates metastatic carcinoma."

The tracheo esophageal fistula was evidently the result of the perforation of a squamous cell carcinoma of the esophagus.

Radiographic examination (Fig. 75 A) revealed an area of malignant infiltration at the junction of the upper and middle thirds of the esophagus with barium outlining the trachea at about the site of the infiltration. It was interesting to note that a roentgen examination of the esophagus made 5 days later (Fig. 75 B) failed to show any barium within the trachea. Therefore it was believed that the appearance at the time of the first examination was the result of regurgitation of barium from the esophagus by way of the pharynx as



FIG. 76. Marked irregular infiltration of the upper portion of the esophagus produced by a tumor of the thymus.

a result of the difficulty in swallowing. However as shown on the esophagoscopic examination and on autopsy an actual esophageal tracheal fistula existed. One must assume, therefore, that at the time of the second radiographic study the zone of fistulous communication was temporarily occluded in some manner possibly by inflammatory pathology or by a particle of food which had become lodged at the fistula.

A rare type of malignant infiltration of the esophagus is that produced by secondary invasion from a primary lesion external to the esophagus. The following is a case of this nature.

J. R., male, aged 29. For 3 years this patient had complained of a lump in the left side of his neck, which gradually became considerably enlarged. There was no discharge. Caseous material was obtained on aspiration. Esophagoscopy and bronchoscopy revealed constriction of the trachea and of the lumen of the esophagus. A biopsy taken from a small mass in the esophagus revealed a transitional cell sarcoma, which was considered to be secondary to a malignant tumor of the thymus. Following a course of roentgen and radium

therapy, the size of the mass, which previously had grown from the left side across the suprasternal space to involve the right base of the neck, became considerably reduced in size. Partial resection of the mass was carried out. It was found to extend from underneath the left lobe of the thyroid down to the anterior mediastinum. The pathologic report of the resected mass was transitional-cell epithelioma. The wound healed but left a few draining sinuses. A small mass developed in the left supraclavicular region, which appeared like an abscess and yielded a large amount of purulent material after being incised. The next day air and gastric contents passed through the sinus. Apparently, this was an esophageal-tracheal fistulous sinus.

Roentgen examination of the esophagus (Fig 76) revealed marked infiltration of the upper portion, with numerous irregularly outlined translucent areas apparently secondary to the invasion of the tumor of the thymus.

As a rule, malignancy of the esophagus is of primary origin. Metastatic spread to the esophagus from a primary carcinoma in a distant location is very rare. Von Recklinghausen¹ described a myxochondrosarcoma of the tibia which showed generalized metastases associated with implantation in the esophagus. Spiegelberg's case² was a melanomasarcoma of the left eyeball, followed by death 13 months after enucleation. In addition to generalized metastases, the autopsy disclosed implantations in the esophagus.

Gross and Freedman³ described a case of an obstructing tumor of the esophagus which was due to a metastatic implantation from a primary carcinoma of the prostate.

Toreson⁴ described the findings in three patients autopsied at the Pathological Institute of McGill University in which metastatic carcinoma of the esophagus was found originating in a primary lesion elsewhere. In two cases, the primary lesion was a carcinoma of the breast, in the third case, a histoid teratoma with embryonal carcinoma of the testis. All three patients had developed dysphagia before death, and the autopsies showed that metastatic lesions had occluded the esophagus.

Differential diagnosis on the basis of the

roentgen evidence between a primary lesion and one of metastatic origin cannot be made.

Rarely, a carcinoma may develop at the site of a benign stricture of the esophagus. Benedict,⁵ in a survey of the literature, found reports of 31 cases of carcinoma developing at the site of an originally benign stricture of the esophagus. To these he added 2 cases of his own. Of all these cases 16 occurred in strictures which had resulted from lye.

Sarcoma of the Esophagus Sarcoma of the esophagus is rare. Chapman in 1877⁶ reported the first case of a tumor which had occluded the esophagus and at autopsy proved to be an oval and spindle-celled sarcoma. The spindle-celled sarcoma reported by Targett in 1888⁷ is preserved in Guy's Hospital Museum and is the first example of this type of lesion recorded in the transactions of the Pathological Society of London. Although the number of reported cases has gradually increased, sarcoma of the esophagus may still be considered a very rare disease. The most unusual type is the leiomyosarcoma first reported by Howard.⁸

As already noted (page 102), sarcoma of the esophagus may simulate the appearance grossly and roentgenologically of a benign, intraluminal, polypoid tumor and even be possessed of a pedicle. In many cases, however, the deformity produced by the lesion is indistinguishable from that of carcinoma.

The spindle-cell sarcoma reported by Tanew⁹ was found at autopsy to be a polypoid tumor with a pedicle attached to the left pyriform sinus. However, the roentgen deformity produced by the tumor was very irregular and not distinguishable from a carcinoma.

Similarly in the case reported by Cottet¹⁰ of a sarcoma of the esophagus, the roentgen deformity was indistinguishable from a carcinoma, although the resected specimen was polypoid and intraluminal.

In the rhabdomyosarcoma of the esophagus reported by Thorek and Neiman¹¹ the

radiographic appearance was also indistinguishable from an infiltrating carcinoma. An extremely rare type of tumor is the primary melanoma of the esophagus described by Burnett and St John.¹⁷ The roentgenogram showed evidence of an obstructive neoplasm at the cardiac end of the esophagus. There were no distinguishing features. The nature of the tumor was discovered on histologic examination of the resected specimen. There were only 4 other cases of a melanoma of the esophagus reported in the literature.

The following is a case of sarcoma of the esophagus.

Illustrative Case P. F. male aged 66. The patient gave a 7 months' history of dysphagia at first limited to solid foods but later extending even to liquids. He had lost 45 pounds during this time.

Esophagoscopy revealed a sloughing mass on the posterior wall involving both lateral walls of the esophagus about the middle third. There was almost complete obstruction of the lumen.

Pathologic examination of a biopsy specimen removed through the esophagoscope was reported as follows: Microscopic examination shows pieces of tissue composed of a tumor mass which consists of oval round and spindle shaped cells, the latter predominating. The cells show different staining properties, the majority are hyperchromatic. The tumor cells are embedded in a connective tissue stroma which contains numerous giant cells and many mitotic figures and eosinophiles. Diagnosis: fibrosarcoma.

Roentgen examination (Fig. 77) showed a marked irregular narrowing of the esophagus



FIG. 77. Fibrosarcoma of the esophagus.

at the junction of the upper and middle thirds with almost complete obstruction in this region and secondary dilatation above the area of involvement.

From the roentgen appearance one could state only that a malignant lesion was present and therefore in all probability that the pathologic process was a carcinoma. There are no features in the behavior of this lesion which might have led to the suspicion that the tumor was a sarcoma. The diagnosis depended entirely upon the histologic characteristics of the new growth.

REFERENCES

1. Von Recklinghausen F. Über die venöse Embolie und den retrograden Transport in den Venen und in den Lymphgefäßen. *Virchow's Arch. path. Anat.* 100: 508, 1883.
2. Spiegelberg H. Drei seltene Sectionsbefunde. *Virchow's Arch. path. Anat.* 142: 553, 1895.
3. Gross Paul and Freedman Lester J. Obstructing secondary carcinoma of the esophagus. *Arch. Path.* 33: 361, 1942.
4. Toreson W. E. Secondary carcinoma of the esophagus as a cause of dysphagia. *Arch. Path.* 38: 82, 1944.
5. Benedict E. B. Carcinoma of the esophagus developing in benign stricture. *New England J. Med.* 224: 408, 1941.
6. Chapman S. H. Sarcoma of inferior constrictor of pharynx and inlet of esophagus. *Am. J. M. Sc.* 74: 433, 1877.
7. Targett J. H. Sarcoma of esophagus. *Tr. Path. Soc. London* 40: 76, 1888, 1889.

- 8 Howard, W T , Jr Primary sarcoma of the esophagus and stomach, J A M A 38 392, 1902
- 9 Tanew N Beitrag zur Diagnose der polyposen Oesophagustumoren, Wien klin Wchnschr 59 745 1947
- 10 Cottet, Pierre Tumeurs exceptionnelles de l'oesophage sarcoma polypeux et fibrome stenosant, Radiol clin 18 258, 1949
- 11 Thorek, P , and Neman, B H Rhabdomyosarcoma of the esophagus, J Thoracic Surg 20 77, 1950
- 12 Burnett, J M , and St John, E Primary melanosarcoma of the esophagus, Radiology 57 868, 1951

Foreign Bodies in the Esophagus

ROENTGEN DIAGNOSIS

According to Paluçvay, Hochenegg was the first (1896) to demonstrate the value of localization of foreign bodies in the esophagus by means of the roentgen ray. He also employed fluoroscopic visualization as a guide in the removal of such foreign bodies.

Iglauer and Ransohoff¹ first called attention to the presence of air in the cervical region at the site of perforation of the esophagus by a foreign body. Iglauer suggested the administration of a small quan-

tity of Lipiodol. A streak of oil beyond the confines of the esophagus itself speaks for perforation.

McGibbon and Mather³ reported two cases in which, as a result of the perforation, there was a collection of air which displaced the esophagus anteriorly. Both the presence of the air, as well as the displacement of the esophagus produced by it, could be demonstrated in the roentgenogram. The depth between cervical vertebrae and trachea is increased.

Nonopaque foreign bodies commonly encountered are chicken bones, buttons of a



FIG 78 (Left) Cup shaped defect due to obstruction of the esophagus by food



FIG 79 (Right) A rounded translucent, intraluminal shadow produced by a piece of lamb chop

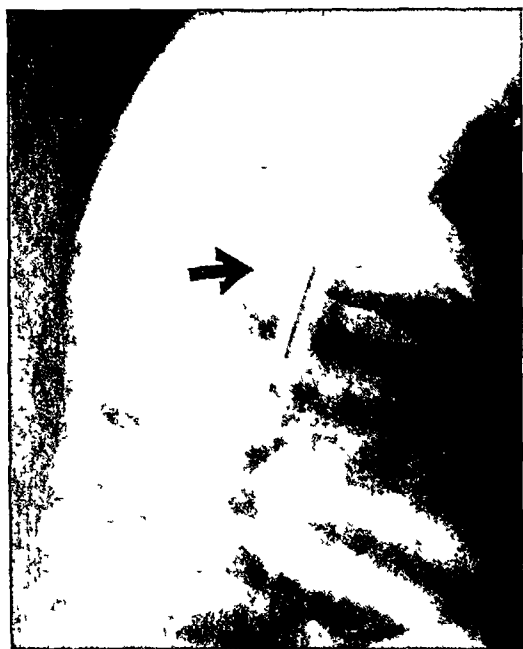


FIG 80 Safety pin in the esophagus

radiolucent character or pieces of meat. They are usually lodged at the suprasternal notch. In some cases, a splitting of the stream may be noted with a thick suspension of barium sulfate in water, or, if the occlusion is marked, there may be an arrest of the flow of barium. After most of the barium has left the esophagus in those cases in which obstruction is only slight, there may be a residual fleck of barium sticking to the foreign body. Manges⁴ was able to demonstrate the thread holes in buttons after administering barium by the distribution of the barium over them.

Scott and Moore⁵ recommended a very thin suspension of barium in water, so as to diminish considerably the opacity of the esophagus after complete filling. Through this thin layer, the filling defect produced by the foreign body may be seen.

A foreign body sometimes may be demonstrable after the administration of a barium-containing capsule, which is arrested at the site of obstruction.

Illustrative Case An example of the de-

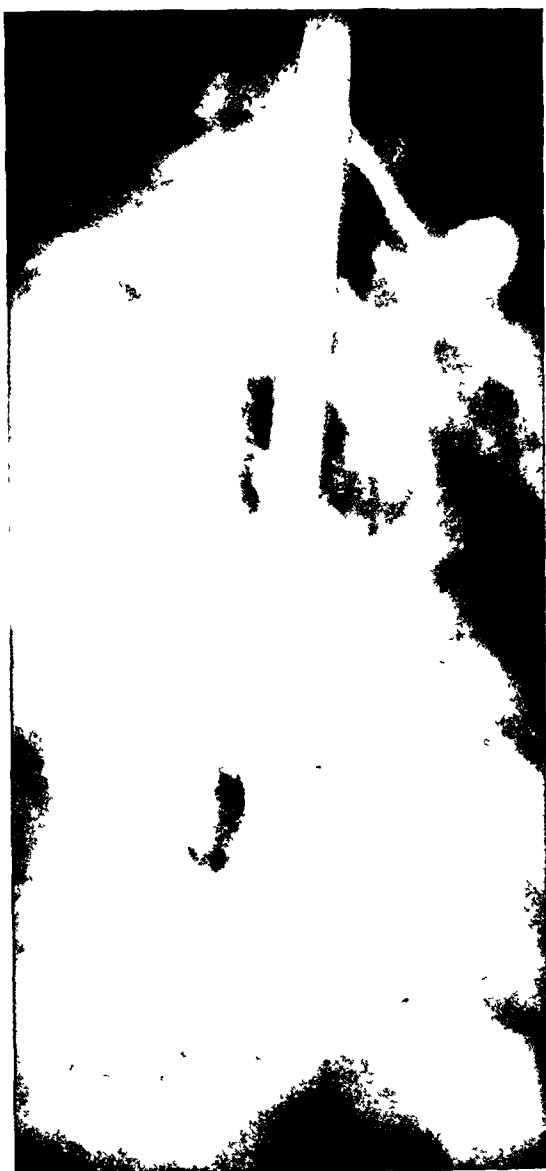


FIG 81 Sword in the esophagus

formity produced by the impaction of food within the esophagus is shown in the following case.

J. C., male, aged 55. This patient had a stricture of the esophagus as the result of having accidentally swallowed a solution containing lye 6 years before his admission to the hospital. Gastrostomy was done because of the esophageal stricture. He had been dilating this stricture of the esophagus for 2 years.

preceding admission. Two days before admission, he found that he was unable to dilate the stricture and was unable to swallow food. This occurred following a meal. The patient said that he had had in the past similar attacks of difficulty in swallowing as a result of food having become lodged in the esophagus above the stricture. This patient made a prompt recovery and within 3 days was able to swallow food.

Roentgen examination (Fig. 78) revealed a translucent area at about the midportion of the esophagus with a cup-shaped rounded defect intraluminal in position preventing the barium above it from passing distally. The smooth rounded intraluminal character of the shadow is characteristic of a foreign body causing obstruction.

Figure 79 shows the irregularly mottled translucent area produced by a piece of lamb chop stuck in the esophagus.

Almost any object may be stuck in the esophagus. Figure 80 represents a safety pin. An unusual example of a foreign body is shown in Figure 81, a sword introduced by the patient and occupying the entire length of the esophagus.

An unusual foreign body in the esophagus is shown in Figure 82. The patient, A. D., female, aged 16, a psychotic, swallowed two coiled bed springs. Radiographic examination shows one of the coiled bed springs in



FIG. 82 Note the two coiled bed springs, one in the esophagus and one in the stomach.

the esophagus and the other in the stomach. One bed spring was removed by way of the esophagoscope and the other through a gastrostomy opening.

REFERENCES

1. Iglaue, S. and Ransohoff, J. L. Perforation of the esophagus by a foreign body with a report of a case presenting unusual x-ray signs. *Laryngoscope* 34: 821, 1924.
2. Iglaue, S. Discussion on H. B. Orton. Mediastinitis following esophageal foreign body. *Arch. Otolaryng.* 12: 846, 1930.
3. McGibbon, J. F. and Mather, J. H. Perforation of the esophagus by swallowed foreign bodies, with radiological notes, *Lancet* 2: 593, 1935.
4. Manges, W. F. The roentgenology of foreign bodies in the esophagus and gastrointestinal tract, *S. Clin. North America* 14: 89, 1934.
5. Scott, W. G. and Moore, S. A method of roentgen diagnosis of nonopaque foreign bodies in the esophagus, *J. A. M. A.* 106: 906, 1936.

Esophageal Varices

A knowledge of the venous drainage of the esophagus is essential in order to understand the genesis of varices in this region.

In the submucosa of the esophagus there is a venous plexus from which branches pass through the muscle to an intercommunicating venous plexus on the external surface of the esophagus. From the upper part of this plexus, the blood drains to the inferior thyroid veins, which, in turn, empty into the innominate veins. From the middle portion the drainage is to the azygos vein, which empties into the superior vena cava. There is also some drainage to the hemiazygos vein, but this in turn empties into the azygos. From the lower part of the esophageal plexus, the drainage is to the esophageal branches of the coronary vein, a tributary of the portal vein. This, then, is of great importance, as it constitutes a communication between the portal circulation and the venous circulation of the esophagus. Obstruction of the venous circulation, such as might occur in cirrhosis of the liver, results in a shunting of the blood from the portal system up through the coronary vein and esophageal plexus to pass via the azygos vein into the superior vena cava. Of course, the esophageal veins must dilate under this load, which, if excessive, will lead to the formation of varices. The greater the portal obstruction, the higher up on the esophagus the varicosities are likely to extend. It has been noted that in cases of thrombosis of the splenic vein, esophageal varices may also form. This is apparently due to the fact that the short gastric veins pass up from the splenic vein to the cardiac end of the

stomach, and there a communication with the esophageal veins takes place. It is thus apparent that thrombosis of the splenic vein would result in a back flow of blood along the short gastric veins to the stomach and thence up to the esophageal plexus.

The first examples of the roentgen appearance of varices of the esophagus were those published by Wolf.¹ He employed a thin suspension of barium and demonstrated rounded, circumscribed, translucent areas on the posterior wall of the esophagus produced by the displacement of the barium resulting from the presence of varices. The patient died of hemorrhage. Autopsy revealed syphilitic involvement of the liver and spleen. In the distal third of the esophagus varices were present, confirming the roentgen findings. Wolf made the following additional observations of a fundamental nature. The roentgen evidence of varices may disappear for a time, only to recur in the identical area with an identical appearance. There may be associated delayed emptying of the esophagus. Such varices may be demonstrable before there is any clinical evidence of cirrhosis of the liver. Examination of the esophagus for varices is particularly indicated when there is bleeding of unknown origin.

The validity of the roentgen findings in the diagnosis of esophageal varices was verified in the reports by Hjelm,² Schatzki³ and Kirklin and Moersch.⁴ Of considerable interest were the findings reported by Brdiczka and Tschakert.⁵ In one of the cases, controlled by autopsy, the veins in the specimen were distended with gelatin. The esophagus was then filled with contrast

substance and radiographed. The appearance in the roentgenogram was similar to that obtained during life.

ROENTGEN DIAGNOSIS

In the examination of the esophagus for the presence of varices, a mixture of equal parts of barium and water is employed, rather than the thick barium-acacia mixture ordinarily recommended for the demonstration of organic lesions. This is important because the thick paste may obscure the presence of varices, particularly when they are small and few in number. A large quantity of the barium suspension should be avoided since it may not only obscure varices which are present, but in addition by its mechanical effect it may squeeze the blood out of the distended veins and make their recognition difficult or impossible.

The thin layer method with observation when most of the barium has left the esophagus may permit the varices to appear more clearly as translucent areas. If the barium-water mixture runs through the esophagus too rapidly to permit proper visualization of the lesion, then a barium-acacia mixture as previously described, may be substituted. In some cases, however, owing to an associated obstruction at the cardiac end of the esophagus, the simple barium-water mixture will be retained sufficiently long for detailed study of the varices to be made.

While visualization of the lesion may be made during examination of the patient in the erect oblique position, an additional aid may be examination with the patient prone. The esophagus is observed fluoroscopically through various angles of obliquity during the course of the passage of the barium suspension into the stomach. Films are then taken promptly, after determining the degree of obliquity and the amount of barium filling best suited for their demonstration.

The best time for the exposure to be made is during inspiration. The thoracic veins are more distended at such times and

clearer evidence of the varices may then be obtained.

✓ The first roentgen evidence in the early stage of the formation of varices consists of a widening of the mucosal folds as a result of the venous engorgement. As the individual veins enlarge, varices develop which elevate the mucosa and protrude into the lumen. As these varices gradually increase in size, the circumscribed, rounded defects characteristic of the condition are produced in the roentgenogram. The earliest manifestations of these rounded, translucent areas are to be found at the lower end of the esophagus. As the venous congestion continues, more and more of the esophagus takes part in the process and, in unusually advanced cases, the entire thoracic esophagus, and rarely even the cervical region, may be involved.

In some cases of varices at the lower third of the esophagus, the extreme distal end present in the abdomen may be free of such evidence. In other cases, however, even this region shows definite evidence of their presence.

It is possible that the occasional failure of varicosities to be visualized in the region of the diaphragm is due to the fact that the latter, by exerting pressure upon the esophagus, may express the blood from these vessels so that they cannot be seen.

In addition to the small rounded, beaded areas, intraluminal in position and occupying as a rule the distal portion of the esophagus, there may be abnormally prominent longitudinal folds, apparently representing swollen portions of the veins (free, however, of actual varicosities at the moment of examination).

Besides the alterations of the mucosal relief within the lumen, the contour of the esophagus itself may also be irregular in outline as a result of the formation of varices.

There may be obstruction at the cardiac end of the esophagus, with delay in the exit of the barium and moderate secondary dilatation. The most likely cause is the

actual mechanical presence of the varices themselves, which partially occlude the lumen

A comparison of different exposures made in rapid succession may show considerable variation in the appearance produced by the varices. Such inconstancy of appearance, far from indicating that no real organic disease is present, is evidence that actually favors the diagnosis of varices for the following reasons. The presence or absence of these translucent, beadlike areas depends on the degree of engorgement of the blood vessels at the time of examination. This will be influenced by the peristaltic activity of the esophagus. Small varices may be completely stripped of their blood during peristalsis and therefore may fail to show at that particular moment. In later stages, peristalsis may strip only the varices in the upper portion of the esophagus. As the blood enters the lower varices, these may become markedly distended and may show in an even more exaggerated form. Therefore considerable change in the roentgen appearance of esophageal varices may take place in different observations over a period of a few seconds.

As already noted, the degree of distention of the thoracic veins will vary with the respiratory phase, so that roentgenograms taken during inspiration will show more pronounced manifestations of their presence. If exposures have also been made during expiration, this will be a factor in causing a variation in the relative prominence of the roentgen findings. And, as previously stated, the ingestion of a large amount of barium may also force blood from the varices and therefore be another factor in causing variations in their relative prominence.

In some cases, an examination may be made for suspected varices after hemorrhage has occurred. Failure to find roentgenologic evidence at this time does not rule out their presence but may mean that the exsanguinating process has emptied the varices in such a way that they no longer protrude

into the lumen, with the production of their characteristic pattern of the relief picture.

Clinically, the demonstration of esophageal varices may be of considerable significance. They may be demonstrable at a time when cirrhosis of the liver cannot be shown in any other way, either by the presence of ascites or positive liver-function tests. In the case of a tumor of doubtful origin in the region of the spleen, the demonstration of varices in the esophagus would indicate that the tumor is actually splenic in origin. The demonstration of varices may also explain the cause of hemorrhage in an individual exhibiting no frank evidence of disease and in whom examination of the stomach and intestines is roentgenologically negative. A knowledge of the presence of such varices will also guard against the indiscriminate use of the stomach tube.

Essential Roentgen Findings The essential findings in the roentgen demonstration of esophageal varices are therefore

✓1 The presence of rounded, translucent areas, beadlike in appearance and usually involving the distal portion of the esophagus.

✓2 There may be momentary variation in the appearance of these areas primarily as a result of peristaltic activity, that being the most important factor beyond the control of the examining physician.

As a rule there should be no difficulty in differential diagnosis. Trapped globules of air can be recognized readily by their inconstancy and because of the fact that their continuous change in position and their final entrance into the stomach may be noted during fluoroscopic examination.

As a rule, diagnostic differentiation from a malignancy should offer no serious difficulty. In the presence of esophageal varices peristalsis is intact, and the rounded, translucent areas show variations in prominence in different films as a result of those factors which have already been considered. In malignancy, the contour is irregular, rigid and free of peristaltic activity. Any invasion of the lumen by a proliferating



FIG 83 (A, Left) Esophageal varices (B, Left center) Same patient Note the variation in the appearance produced by the varices (C, Right center) Same patient Note further variation in the appearance produced by the varices (D, Right) Same patient Note that the appearance produced by the varices is different in each observation This is characteristic of the deformity produced by esophageal varices

new growth produces irregularly outlined translucent areas ordinarily lacking the beadlike arrangement of varices. Moreover, the appearance is identical in the different films and is not influenced either by peristaltic activity or by variations in the respiratory phase during which exposures have been made.

Illustrative Cases J T, male aged 31 During the previous 1½ days before admission, this patient had been vomiting blood and passing tarry stools. In addition, he had had pain which was sudden in onset and present in the epigastrium and periumbilical region. It was colicky in nature. He had had no previous episodes of this kind.

Physical examination revealed the presence of ascites. The spleen was markedly enlarged. The liver was not palpable. There was a severe hypochromic anemia. The spleen was particularly palpable after the ascites had diminished under treatment with ammonium chloride and mercupurin. It was then palpable five fingers breadth below the left costal margin.

Esophagoscopy examination showed evidence of varices in the distal third of the esophagus.

Roentgenographic examination (Fig 83 A, B, C, D) revealed numerous irregularly rounded, translucent areas intraluminal in location and primarily limited to the cardiac

end of the esophagus. The various films show a marked alteration in the mucosal relief picture due to the momentary changes in the degree of distention of the varices. At times the translucent areas appear as if outlining a tortuous vein in the course of which may be noted small rounded beadlike protuberances. At times also some of the translucent areas appear as sharply punched out defects of contour.

Variations in the mucosal relief of the esophagus are also illustrated by the following case.

D C male aged 70. Three years previously the patient had been admitted to the hospital after having vomited a considerable amount of blood. At that time he gave a history of having had abdominal paracentesis on five occasions. He again had a recurrence of vomiting of blood the following year. One week before admission he developed fluid in the abdomen requiring paracentesis. About 3 quarts of fluid were removed. His abdomen however continued to distend because of the prompt reaccumulation of fluid. He had had a suprapubic cystostomy for carcinoma of the prostate 6 years before.

Physical examination revealed marked abdominal distention due to fluid and a suprapubic tube draining the bladder. On rectal examination there was a large nontender irregular nodular prostate. The patient

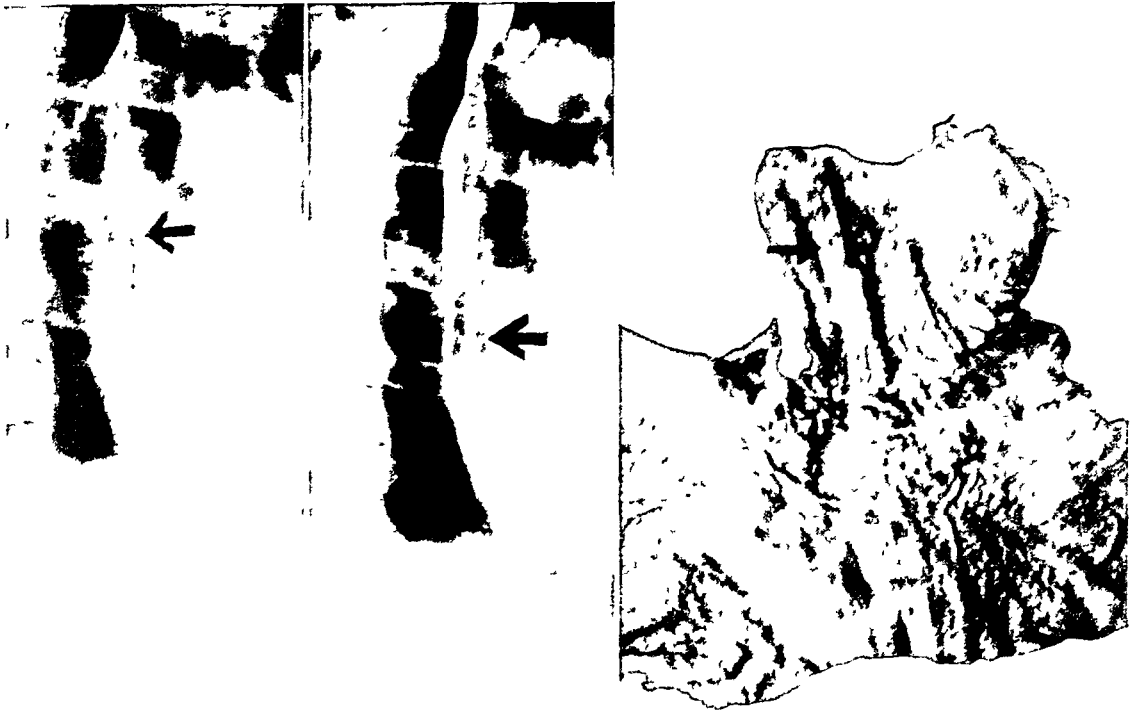


FIG 84 (A, *Left*) Esophageal varices Note the beadlike translucencies in the mid-portion (B, *Center*) Same patient Note the variation in the appearance produced by the varices (C, *Right*) Same patient Autopsy specimen Note the probe inserted into the opening in the vein from which fatal bleeding occurred



FIG 85 (A, *Left*) Esophageal varices Note the translucent areas at the cardiac end (B, *Right*) Same patient Roentgen appearance of the esophageal veins in the autopsy specimen after injection with barium suspension

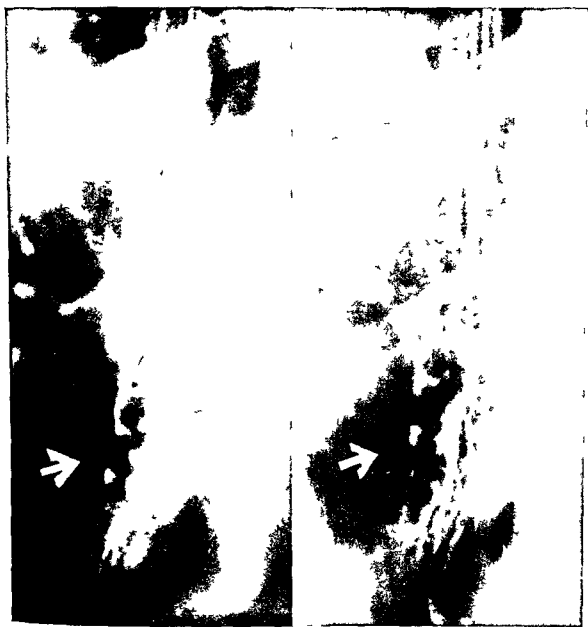


FIG 86 (A, *Left*) Esophageal varices (B *Right*) Same patient Note the changes in the pattern produced by the varices Note also the transition from normal mucosal folds to the distortion produced by the varices

vomited blood on a few occasions while in the hospital and died in spite of transfusion

Autopsy revealed a carcinoma of the prostate with urethral obstruction and a portal cirrhosis of the liver

With regard to the gastro intestinal tract the report was as follows The esophagus presents in its lowermost end several tortuous elevated, linear areas due to underlying varicose veins At several small points small blood clots are noted and pressure on these dilated veins displaces the blood clot and forces blood into the esophagus

During his final brief hospitalization of 8 days before death this patient's physical condition precluded any roentgen studies However roentgen examination of his esophagus had been made 3 years before this last admission

Figure 84 A showed a superficial irregularity in the contour of the distal half, with an elongated translucent area hugging the anterior wall At about its midportion are several rounded beadlike translucencies and proximally an elongated translucent

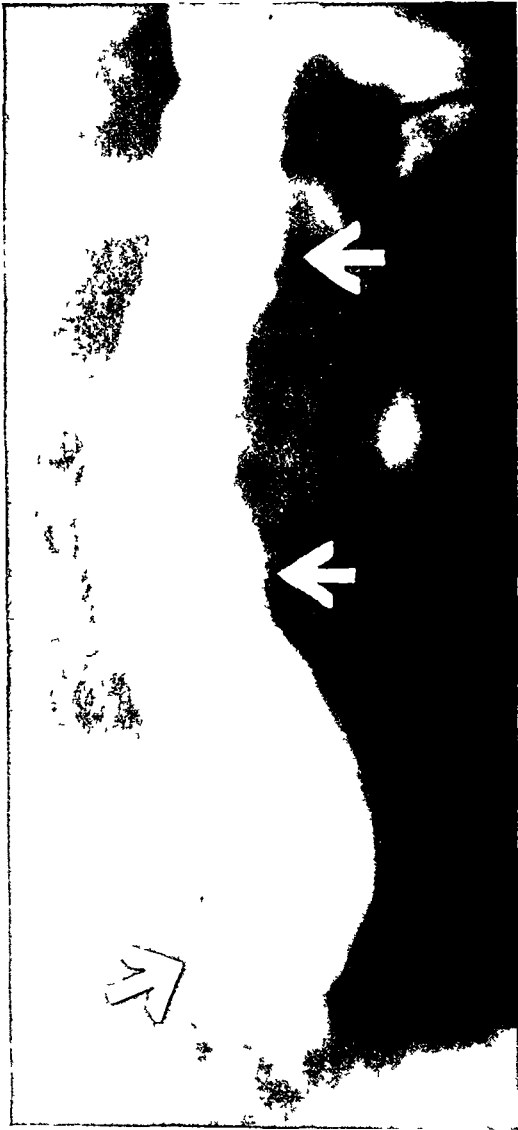


FIG 87 Esophageal varices. Note the dilatation of the esophagus, as well as the translucent areas produced by the varices

area. Figure 84 B showed the appearance of the esophagus at another stage when the distal portion was narrowed and an elongated, centrally located translucent zone was present in this region. The rounded, beadlike translucent areas previously described were again present, although there was a slight change in their configuration. Proximal to this region was a vaguely outlined, elongated, centrally located trans-

lucent zone with very small, rounded areas in its course. The entire picture was that of esophageal varices and explained the clinical symptomatology, the ascites and the vomiting of blood. Figure 84 C showed the appearance at autopsy with a probe through an opening in a distended esophageal vein from which the bleeding had occurred.

Short, elongated areas of translucency with deformity of the contour produced by varices is illustrated by Figure 85 A in the case of S. V. These findings were transient, however, and there was considerable variation during the course of examination. The diagnosis of varices was controlled by autopsy findings. Figure 85 B showed the roentgen appearance of the esophageal veins in the autopsy specimen after injection with a barium suspension.

The transition from normal mucosal folds to the distortion produced by varices is illustrated by the next case.

M. L., female, aged 52. The patient gave a 5-week history of generalized abdominal discomfort, vomiting and moderate weight loss. During the preceding 5 days she noticed a swelling of the abdomen. Physical examination revealed evidence of ascites, enlargement of the liver, engorgement of the cervical veins and moderate ankle edema. She had severe secondary anemia.

The roentgenographic examination (Fig 86 A and B) showed classical changes in the distal half of the esophagus produced by esophageal varices. Figure 86 A showed a beadlike arrangement along the course of the distal portion of the esophagus near its anterior wall. In Figure 86 B considerable change in the deformity is seen, a characteristic of esophageal varices. Rounded translucent areas not shown in Figure 86 A may be noted in Figure 86 B, probably due to the thinner layer of barium. Note also, by way of contrast, the normal parallel arrangement of the mucosal folds in the proximal portion of the esophagus in Figure 86 B.

The esophagus may become considerably dilated because of varices as in the illustrative case that follows.

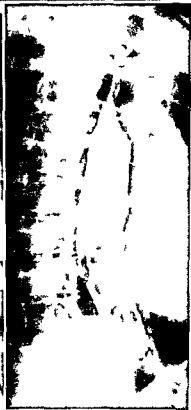
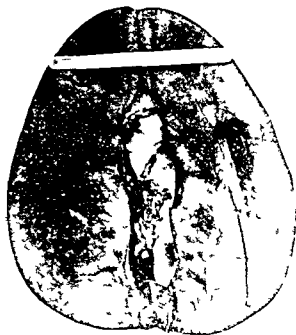


FIG 88 (A *Top left*) Appearance of the spleen removed in a case of esophageal varices (B, *Top right*) Roentgenographic appearance produced by esophageal varices about 3 years after the onset of episodes of massive hemorrhage (C *Bottom left*) Appearance about 1 year later (D *Bottom center*) Appearance 9 months after C (1 year and 9 months after B) (E *Bottom right*) Appearance 8 years and 3 months after D and about 8 years after splenectomy, following which the patient was clinically well without any further recurrence of hemorrhage In spite of this the roentgen deformity persisted

L F, male, aged 55. The patient gave a history of lues and of pulmonary tuberculosis. Three days before admission to the hospital, he developed epigastric pain, which was followed shortly by the vomiting of blood and by tarry stools. He had had no previous gastro-intestinal symptoms. While under observation his abdomen became distended as a result of ascites.

The autopsy revealed portal cirrhosis of the liver in addition to pulmonary tuberculosis. There were marked esophageal varices, which extended all the way up to the pharyngeal region.

The roentgenographic examination (Fig 87) showed marked dilatation of the entire esophagus, with intraluminal translucencies throughout its entire length. There was considerable change in the appearance of these areas in the different films. No other cause but varices was found at autopsy to explain the dilatation. The widespread character of the involvement was also confirmed at autopsy, which showed varices as high up as the pharyngeal region.

The following case of esophageal varices is of interest in that repeated observations were made over a period of 12 years. The patient, N F, female, 47 years old at the time of the last examination, had entered the hospital at the age of 35 with a history of repeated episodes of vomiting of blood and tarry stools. Physical examination revealed an enlarged liver and spleen. She was hospitalized after each hemorrhage, making an uneventful recovery following blood transfusion and antianemic therapy. She was luetic and was being treated actively during this time. The diagnosis was bleeding from esophageal varices. This was based on the clinical history as well as the classical evidence obtained in the roentgenographic examination of the esophagus. Finally, a laparotomy was done. At operation the liver appeared to be normal. The spleen was markedly enlarged with almost complete occlusion of the splenic vein. An omentopexy and a splenectomy were done.

Pathologic examination of the spleen disclosed giant follicular lymphoma. Figure 88 A shows the appearance of the resected spleen, spread open. Examination of the splenic vein showed no evidence of throm-

bosis as it entered the spleen. The roentgen evidence upon which the diagnosis of esophageal varices was based, as well as the changes which occurred in the years before and after operation, are shown in Figures 88 B to E. Figure 88 B represents a characteristic appearance of the esophagus approximately 3 years after the original radiographic examination which disclosed evidence of esophageal varices. Note the two main types of translucencies in the distal portion of the esophagus: (1) the small, rounded, sharply defined translucent areas, apparently representing the actual varices, (2) the elongated rather tortuous translucent area representing the distended vein itself.

Figure 88 C represents the appearance approximately 1 year later, and Figure 88 D the appearance 9 months after Figure 88 C or 1 year and 9 months after Figure 88 B. Following the splenectomy the patient felt better. There was no recurrence of hemorrhage. However, repeated roentgen examinations of the esophagus still showed persistent evidence of the translucent areas produced by the varices. The patient disappeared for 7 years. She then returned, stating that during all this time she had felt well and had had no episode of bleeding.

Radiographic examination at this time (Fig 88 E), 9 years after the examination recorded in Figure 88 B and about 12 years after the original episode of hemorrhage and 8 years after the splenectomy, again showed persistent evidence of translucent areas in the distal segment of the esophagus. Therefore, although the patient was clinically well, there was no apparent regression in the degree of deformity produced by the varices.

ESOPHAGEAL VARICES IN CHILDREN

Roentgen evidence of varices may also be noted in children. In Jorup's⁶ case varices were demonstrated when the child was 6 months old. Hansson⁷ described

esophageal varices in three children. The youngest a boy was 9 months old the other two were aged 3 years and 5 years. Clinical evidence of esophageal varices was found in all three. In one case autopsy confirmed the presence of the varices. There was a thrombosis of the portal and the splenic veins. The possibility of esophageal varices in the newborn is suggested by Vorpahl's observation in 1912. He described the findings at autopsy in a child that died of hemorrhage on the third day

after a normal birth. The blood which filled the stomach and the entire intestine had originated in the esophagus in which under the mucosa were numerous varicosities which could be seen with the naked eye. The extremely thin walled veins were present mainly under the epithelium of the esophagus and were so closely packed that microscopically the appearance simulated a hemangioma. The venae cavae and the liver were normal. (There was no notation about the condition of the splenic vessels.)

REFERENCES

- 1 Wolf G. Die Erkennung von Ösophagusvarizen im Röntgenbilde. Fortschr. Geb. Röntgenstrahlen 37:890, 1928.
- 2 Hjelm R. Zwei Fälle von röntgendignostizierten Ösophagusvarizen, Acta radiol. 12:146, 1931.
- 3 Schatzki R. Die Röntgendiagnose der Ösophagus und Magenvarizen und ihre Bedeutung für die Klinik. Fortschr. Geb. Röntgenstrahlen 44:28, 1931.
- 4 Kirklin B. R. and Moersch H. J. Report of a case of roentgenologically demonstrable esophageal varices complicated by splenomegaly. Radiology 17:573, 1931.
- 5 Brdiczka I. G. and Ichikert J. Die röntgenologische Diagnostik der Ösophagusvarizen. Fortschr. Geb. Röntgenstrahlen 46:156, 1932.
- 6 Jorup Sigvard. Congenital varices of the esophagus. Acta paediatr. 35:247, 1948.
- 7 Hanson C. J. Varices of the esophagus in children. Acta radiol. 25:507, 1944.
- 8 Vorpahl I. Ueber Melena neonatorum. Deutsche med. Wchnschr. 38:245, 1912.

Peptic Ulcer of the Esophagus

The occurrence of ulcer of the esophagus has been recognized for many years. Billard,¹ in 1828, described ulcers of the esophagus in a newborn boy who was unable to retain any nourishment and came to autopsy.

I made the autopsy 6 hours after death.

At the upper part of the esophagus there were two ulcers almost parallel, oblong and about 4 lines in length. The center was yellowish. The edges as well as all of the upper third of the esophagus were a bright red color. The stomach and the intestinal canal were markedly congested, and there was a bloody exudate throughout the entire length of the digestive tract. It is evident that the infant had at birth inflammation and ulceration of the esophagus, a serious affection which must have interfered considerably with swallowing.

One of the earliest fully documented cases of a perforating ulcer of the esophagus is to be found in the report by Flower² in 1853. The patient was a male, aged 51, who had complained of substernal pain radiating to the back. Death followed a sudden attack of profuse hemorrhage from the mouth. At autopsy in the esophagus 3 inches above the cardia there was "a perfectly circular opening $3\frac{1}{2}$ lines in diameter with slightly elevated edges as if cut out with a punch, exactly resembling on a small scale the circular perforating ulcer so frequently met with in the stomach." Examination revealed perforation leading from the ulcer into the aorta, which was the cause of the fatal hemorrhage.

Knott³ also quoted in detail a case of an ulcer of the esophagus reported by Mr. Salter in 1853 before the Royal Medical and Chirurgical Society of London. In that

case the ulcer had perforated into the left bronchus. He also included a case reported by Floriner of an esophageal ulcer that had perforated into the aorta.

Another early report of a case of perforation of an ulcer of the esophagus is that presented by Gordon in 1855 before the Pathological Society of Dublin.⁴ The specimen showed a chronic oval ulcer of the esophagus $\frac{1}{2}$ inch in extent at about the level of the bifurcation of the trachea, which exhibited a fistulous communication with an abscess of the left lung.

Benson,⁵ in 1873, presented a specimen before the Dublin Pathological Society of a perforating idiopathic ulcer of the esophagus removed from the body of a woman, aged 50, who during life had complained of extreme dysphagia and soreness referred to the center of the sternum. Autopsy disclosed an oval opening in the esophagus about 5 inches proximal to the cardiac end caused by an ulcer that had perforated through all the layers, including the layer of pleura covering it.

Among the earliest contributions to the study of peptic ulcer of the esophagus was that of Quincke,⁶ who in 1879 described three cases. These lesions were present in the distal portion of the esophagus and, in his opinion, had been produced by the action of regurgitant gastric juice. In his third case stenosis resulted from the action of the ulcer.

The condition had been considered extremely rare by Rokitsansky, and its existence had been completely denied by no less an observer than Birch-Hirschfeld. Quincke, however, dissipated all doubt by his histologic studies.

At the time of the publication of his paper in 1906, Tileston found records of 41 cases of peptic ulcer of the esophagus described in the literature. To these cases Tileston added his observations in 3 cases of his own. A study of the records of the Massachusetts General, the Boston City, and the Long Island Hospitals disclosed 6 cases of peptic ulcer of the esophagus in a series of 4,496 autopsies, an incidence of 0.13 per cent. In the Bellevue Hospital series of 22,810 autopsies from 1905 through 1935, there were 30 cases of ulcer of the esophagus, or an incidence of 0.13 per cent. Strangely enough, this figure is identical with that of Tileston. The probable accuracy of this relative incidence of peptic ulcer of the esophagus is enhanced by the fact that it holds for the much larger autopsy series at Bellevue Hospital.

Ewald⁸ in 1910 described stenosis of the esophagus resulting from the presence of a peptic ulcer.

Another important contribution to the study of peptic ulcer of the esophagus was that of Cantieri.⁹ In his monograph he made a thorough survey of the literature up to 1910 and credited Wolcamero with having recorded the first case in 1671. Cantieri collected reports of 62 cases and added 6 of his own, including the actual pictures of the specimens showing the presence of the ulcer of the esophagus.

Müller in 1912¹⁰ described an ulcer of the esophagus just above the cardiac orifice about $1\frac{1}{2}$ inches in its greatest diameter which had perforated into the left mediastinum and left pleura. A duodenal ulcer was also present.

Watson in 1912¹¹ described two cases of ulcer of the esophagus found at autopsy at St. Bartholomew's Hospital. In both cases the ulcer was situated immediately above the cardiac orifice. One had perforated into the left pleura. In Roszak's case¹ autopsy in a 2 year old child revealed a round ulcer of the esophagus with perforation into the left pleural cavity. Perforation of two ulcers at the lower end of the esophagus found at

autopsy in a patient who died of broncho pneumonia was reported by Gott.¹³

Simple ulcer of the esophagus resembles that of the stomach, both grossly and microscopically. It is usually found just above the cardia. Ulcers vary greatly in size, ranging from those the size of a pea or smaller to those involving the entire surface from the bifurcation to the cardia. As a rule only one ulcer is present, rarely the condition is multiple. The small ulcer may involve only the mucosa, or, in the more chronic and older form, it may have the typical punched out appearance of a gastric ulcer. There is a great tendency to perforation. Healing may take place with scar formation, with puckering and radiation or with actual stenosis.

A significant factor which may explain the etiology of such peptic ulcers is the presence of aberrant gastric mucosa in the wall of the esophagus. This was demonstrated by Johanna Hellmann,¹⁴ Taylor,¹ Jackson,¹⁶ and Stewart and Hartfall.¹⁷

Kelly¹⁵ examined the esophagus histologically in 17 children under 1 year of age and found islets of gastric mucosa in some of these cases. In one infant 10 weeks old such misplaced gastric mucosa was found in association with an esophageal ulcer just above the level of the diaphragm. There were marked chronic inflammatory changes with early fibrosis as far up as the bifurcation of the trachea.

Rector and Connerley¹² examined the esophagus in 1,000 consecutive autopsies of infants and children and found aberrant gastric mucosa in 118 cases. Inflammation with and without ulceration was frequently present in this heterotopic mucosa. An interesting observation which they made is that in 51 per cent the aberrant gastric mucosa was in the upper third of the esophagus, in 41 per cent in the middle third and in only 8 per cent was it present in the lower third. Acid gastric secretion from such aberrant glands may therefore underlie the formation of the full blown ulcer.

Another factor may be a retrograde flow

of gastric juice up into the distal portion of the esophagus. In some cases peptic ulcer has been noted at the junction of a congenitally short esophagus with a thoracic stomach. The acid gastric secretion within the thoracic stomach by regurgitation may be a definite factor in the explanation of such esophageal ulceration.

A rare cause of peptic ulcer of the esophagus is cerebral or cerebellar disease. Cushing²⁰ described the case of a child of 10 operated on for a cerebellar tumor. At autopsy there was a perforated lesion in the esophagus about 3 cm in length. In another case, following extirpation of a right parietal metastatic hypernephroma, autopsy disclosed both gastric and esophageal perforation.

Evidence of perforation of the esophagus as noted at autopsy in a patient suffering from acute encephalitis was described by Masten and Bunts.²¹

ROENTGEN DIAGNOSIS

The outstanding feature in the roentgen diagnosis of peptic ulcer of the esophagus is the demonstration of the niche. Associated with this there is usually an area of stenosis at the site of the niche. The lesion usually involves the distal portion of the esophagus.

Apparently, the first roentgen demonstration of peptic ulcer of the esophagus was by Brunetti.²² The diagnosis was based on the presence of a large niche in the lower third of the esophagus. This was accompanied by spasm of the esophagus in the region of the niche during the swallowing of a bolus of food. There was a history of recurrent hemorrhage, although no lesion other than the one in the esophagus was demonstrable. The patient apparently died of perforation. Unfortunately, no autopsy confirmation was obtained.

Fleischner²³ described a case in a man 64 years of age, who gave a 5-year history, suggesting gastric ulcer. Roentgen examination showed two niches of the esophagus

just above the diaphragm. The longitudinal folds radiated toward them. About ½ year later, an irregularly outlined sacculation was present, 5 mm by 15 mm in size, at the location of the previous niches. The patient at this time complained of vomiting and of pain upon ingestion of liquid as well as of solid food. A gastrostomy was performed for relief. One month later, roentgen examination showed that the sacculation had completely disappeared and that the contour of the esophagus was normal. The gastric fistula was then permitted to heal. Roentgen examination later showed the esophagus to be persistently normal.

In Aurelius²⁴ case roentgen examination showing a nichelike area at the lower end of the esophagus was confirmed by esophagoscopy. At this examination a small ulcer at the cardiac end of the esophagus was found. A biopsy was taken. Microscopic examination revealed glands resembling those of the stomach. An edge of the section resembled the appearance of a peptic ulcer.

Additional examples of the niche in the diagnosis of peptic ulcer of the esophagus appear in the contributions of Chaoul and Adam,²⁵ Friedenwald, Feldman and Zinn,²⁶ and Mellins.²⁷

A further discussion of ulcer of the esophagus and particularly its relation to esophagitis, will be found in the next chapter.

Differential Diagnosis Differential diagnosis of peptic ulcer of the esophagus from a new growth is based on the following:

- 1 In new growth of the esophagus there may be an area of irregular narrowing without any projection beyond the confines of the contour of the esophagus itself, or the lesion, if of a proliferating character, may produce irregular translucent areas by direct intraluminal invasion. In peptic ulcer of the esophagus the contour as a rule is smooth in character, and the ulcerating lesion projects beyond the confines of the esophagus itself.

- 2 In malignant infiltration of the esoph-

agus there is apt to be a destruction of the normal mucosal markings. When the mucosal structure of the narrowed area of the esophagus in peptic ulcer is demonstrable, it may exhibit no destruction of the normal mucosal folds.

3 In the case of ulceration of a new growth projecting into the mediastinal tissues, the area of ulceration will be of irregular configuration and will be associated with other evidence of infiltration in that particular segment of the esophagus in which ulceration has arisen.

4 New growth of the esophagus is a progressive disease with gradual extension of the malignant process. In the case of a niche of a benign ulceration, roentgen evidence of the lesion may disappear after medical management.

5 Esophagoscopy and biopsy help definitely to differentiate the two disorders in the event of any doubt.

A peptic ulcer of the esophagus may also be differentiated readily from a diverticu-

lum. The latter is more apt to occur on the anterior wall of the esophagus at its mid portion near the bifurcation of the trachea. The diverticulum may be tent shaped or perfectly well rounded and is practically never associated with an area of stenosis. At times the niche may appear to be separated from the rest of the esophagus, as a result of inflammation, edema and spasm. This does not occur in the case of a diverticulum, the neck of which, as a rule, is clearly demonstrable.

In a borderline case, the therapeutic test may be of considerable help, in that while the niche of a peptic ulcer of the esophagus may disappear under medical management, the diverticulum remains identical in size and shape under such treatment. Finally, as has been said, esophagoscopy will aid in differential diagnosis if any doubt still exists. Such a possibility may arise in the case of the perforation of the diverticulum into the mediastinal tissues.

Illustrative Cases The following case

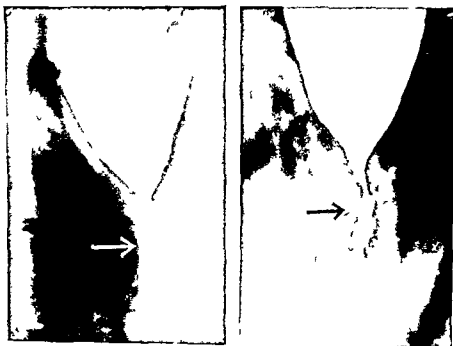


FIG 89 (A *Left*) Peptic ulcer of the esophagus (first oblique position). Note the narrowing in the distal portion of the esophagus with a niche. Note also the dilatation of the esophagus above the constricted region. (B, *Right*) Second oblique position. The niche is more prominent at this time.

shows roentgenographic evidence of peptic ulcer of the esophagus

B R, male, aged 50 This patient gave a history of about 1 year of pain in the epigastrium and of difficulty in swallowing There were intermittent attacks of nausea and vomiting, without blood He had lost 20 pounds since the onset He had a chronic cough, and the sputum at times was blood-tinged

Esophagoscopic examination revealed an area of constriction in the distal esophagus, with a flat ulcer

Microscopic study of a specimen removed through the esophagoscope revealed a diffuse infiltration of lymphocytes and plasma cells

Roentgen examination (Fig 89 A and B) revealed the presence of a smooth area of constriction in the distal portion of the esophagus Beyond this narrowed area was a niche typical of an ulcer in this region In view of the roentgenographic appearance, the esophagoscopic confirmation, the biopsy report of chronic inflammation and the fact that there was no evidence of malignancy, this case may



FIG 89 C Same patient as shown in Figure 89 A and B The appearance of the esophagus after dilatation of the stricture and gastrostomy

be considered as one of peptic ulcer of the esophagus

Following gastrostomy, as well as dilatation of the strictured area, there was a remarkable improvement in the condition, as noted in the roentgen appearance (Fig 89 C)

Figure 90 shows a niche at the distal end of the esophagus This region is narrowed but the mucosal structure is intact A diagnosis of peptic ulcer of the esophagus was made, which was confirmed by esophagoscopy The narrowed area was dilated successfully, and later esophagoscopy showed that the ulcer had disappeared About 2½ years later the patient was clinically well.

The association of an ulcer of the esophagus with herniation of the stomach is illustrated by the following case

S G, female, aged 52 The patient gave a 2-week history of severe right upper quadrant pain, radiating to the back and not definitely related to food She vomited coffee-ground material and had a tarry stool Physical examination of the abdomen was essentially negative She had marked secondary anemia There was no evidence of weight loss In spite of transfusions, she had recurrent massive bleeding A left transthoracic resection was done for vagotomy There was complete fibrosis of the pleural cavity The proximal portion of the stomach was herniated through the esophageal hiatus An ulcer was found to have perforated into the lower end of the esophagus Closure of the perforation was unsuccessful because of the necrosis, friability and extent of ulceration The ulcerated portion of the esophagus was resected, and an esophagogastrostomy was done The surgeon described the esophagus as shortened, probably congenitally

Pathologic diagnosis of the resected specimen was reported as follows "extensive ulceration of the mucosa The ulcer base contains some fibrinoid material and connective tissue infiltrated by chronic inflammatory cells The fibrous tissue extends into the submucosa, and the muscularis partially interrupted and replaced by fibrous tissue The periesophageal tissue is thickened by connective tissue No evidence of tumor is seen Diagnosis Chronic ulcer of esophagus"

Roentgen examination, in addition to disclosing a duodenal ulcer, showed some interesting features in the appearance of the

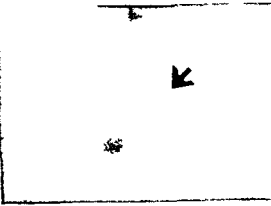


FIG 90 (Left) Peptic ulcer of the esophagus



FIG 91 (Right) Peptic ulcer of the esophagus. The arrow points to the niche in the narrowed segment of the esophagus at its junction with the herniated portion of the stomach

esophagus (Fig 91) The distal end was narrowed at its junction with a supradia phragmatic portion of the stomach. At the strictured zone there was a cone shaped niche. The diagnosis was peptic ulcer of the esophagus. While it was obvious that the esophagus was shortened I was not certain whether this was the result of a congenital anomaly or was

secondary to the stricture causing shortening, with a consequent pull on the stomach, drawing it up into the chest. The surgeon thought the shortening was congenital. Except for the bleeding, which may have originated from the ulcer of the esophagus, there were no clinical symptoms to suggest a lesion in this region. It is also noteworthy that the patient had a duodenal ulcer which might have explained the massive hemorrhage.

REFERENCES

- 1 Billard C. *Maladies des nouveaux nes* observation 21, p 276, Paris 1828
- 2 Flower W H. A case of perforating ulcer of the oesophagus which caused death by penetrating the aorta. *Medico Chir Tr* London 36 353, 1853
- 3 Knott John F. *Pathology of the Esophagus* Dublin, Fannin 1878
- 4 Gordon Samuel. Ulceration of the oesophagus. Fistulous communication with left lung pneumo thorax and acute pleuritis. *Dublin Hosp Gaz* 2 10 1855
- 5 Benson Hawtrey. Case presentation. *Dublin JI M Sc* 56 69 1873
- 6 Quincke H. *Ulcus oesophagi ex digestionem*. *Deutsches Arch klin Med* 24 72, 1879
- 7 Tileston W. Peptic ulcer of the oesophagus. *Am J M Sc* 132 204 1906
- 8 Ewald C A. *Diagnose und Behandlung des Ulcus oesophagi pepticum und Ulcus duodenale*. Berlin klin Wchnschr 47 180 1910
- 9 Cantieri C. Contributo allo studio dell'ulcera semplice (peptica) dell'esofago. *Arch per le sc med* 34 439, 1910
- 10 Miller, J. A case of perforated peptic ulcer of the esophagus complicated by pyopneumothorax. *Brit M J* 1 116 1912
- 11 Watson, C G. Two cases of peptic ulcer of the esophagus. *Brit M J* 2 1182 1912
- 12 Roszak St. *Peptisches Geschwur der Speiserohre mit Durchbruch in die linke Pleurahohle*. *Ztschr Kinderh* 43 181, 1927
- 13 Gott R Jr. Spontaneous rupture of the esophagus with a report of four cases. *Am J M Sc* 186 400 1933
- 14 Hellmann, J. *Das Ulcus pepticum oesophagi*. *Beitr klin chir* 115 449 1919
- 15 Taylor A L. The epithelial heterotopias of the alimentary tract. *J Path & Bact* 30 415, 1927

- 16 Jackson, C Peptic ulcer of the esophagus, J A M A 92 369, 1929
- 17 Stewart, M J, and Hartfall, S J Chronic peptic ulcer of the esophagus, J Path & Bact 32 9, 1929
- 18 Kelly, A B Ascending fibrosis of the esophagus and relation to the presence of islets of gastric mucosa, J Laryng & Otol 54 621, 1939
- 19 Rector, L E, and Connerley, Marion L Aberrant mucosa in the esophagus in infants and in children, Arch Path 31 285, 1941
- 20 Cushing, Harvey Peptic ulcers and the hindbrain, Surg, Gynec & Obst 55 1, 1932
- 21 Masten, M G, and Bunts, R C Neurogenic erosions and perforations of the stomach and esophagus in cerebral lesions—report of six cases, Arch Int Med 54 916, 1934
- 22 Brunetti, L Die Röntgendiagnose des Ulcus pepticum oesophagi, Fortschr Geb Röntgenstrahlen 35 750, 1925
- 23 Fleischner, F Die Röntgendiagnose des Ulcus pepticum oesophagi, Wien klin Wchnschr 40 120, 1927.
- 24 Aurelius, J R Peptic ulcer of the esophagus, Am J Roentgenol 26 696, 1931
- 25 Chaoul, H, and Adam, A Die Schleimhaut des Verdauungskansls im Röntgenbild, eine normale und pathologische Röntgenanatomie der Innenwand des Verdauungskansls, Berlin and Vienna, Urban, 1931
- 26 Friedenwald, J, Feldman, M, and Zinn, W F Peptic ulcer of the esophagus, Am J M Sc 177 1, 1929
- 27 Mellins, H Z Esophageal ulcer in infancy, Am J Roentgenol 68 634, 1952

Esophagitis

Zenker and Von Ziemssen gave one of the earliest descriptions of esophagitis. It is a rare affection.

The condition may result from mechanical, thermal or chemical irritation of the esophagus by food or foreign bodies. Inflammation may also result from external factors, such as conditions in the spine, the mediastinum and the lymph nodes. Also esophagitis may be a localized manifestation of a more general disease, as in some of the infectious diseases, acute exanthemata and possibly syphilis. The inflammation may be catarrhal, with superficial erosions, which frequently heal without leaving any scar. Catarrhal inflammation of long duration may lead to hypertrophic thickening of the mucosa, with the development of polypoid processes. In some cases this may also be associated with considerable hypertrophy of the muscular wall of the esophagus, especially when the condition is complicated by dilatation. Actual ulcers of considerable depth may develop; these may be capable of initiating hemorrhage through involvement of large blood vessels. Atony of the entire esophagus may result from a diminution of the contractile function of the esophagus, thus leading to diffuse ectasia.

The authors further described follicular, fibrinous, pustulous, phlegmonous and corrosive types of esophagitis. Ulcers of the esophagus may develop in all these types of inflammation.

Regurgitation of acid gastric secretion may be a factor in the development of some cases of esophagitis.

The probable causal relationship of re-

gurgitation of acid gastric secretion in the production of some cases of esophagitis, as well as ulcer of the esophagus, is indicated by the following observations:

1. The coexistence of these lesions with ulcer of the stomach and duodenum. Jackson,¹ in 1929, in the study of 88 cases of peptic ulcer of the esophagus described the coexistence of gastric ulcer in some of these cases and assumed that the perpetuating factor, resulting in the chronicity of the esophageal lesion in such a case was the regurgitation of the acid gastric secretion.

In two of the cases of esophagitis reported by Vinson there was an associated duodenal ulcer. C. L. Jackson² emphasized even more clearly the not infrequent coexistence of gastric or duodenal ulcer with inflammatory stricture of the esophagus.

In 2 of the 3 cases of acute perforation of the esophagus described by Hertzog and Leighton⁴ with autopsy confirmation there was an associated duodenal ulcer.

In the cases of peptic ulcer of the esophagus illustrated in this book by Figures 91, 94, 97 and 112 there was an associated duodenal ulcer. Since hyperacidity is commonly present, particularly in duodenal ulcer, repeated regurgitation of the acid secretion into the distal portion of the esophagus may ultimately traumatize the mucosa to such a degree that it becomes a prey to the eventual development of esophagitis.

2. Further support for the concept of the etiologic relationship of the gastric secretion to esophagitis is to be found in the contribution by Bartels in a study of 82 cases of esophagitis studied at autopsy. In every case gastric juice was found in the

lower third of the esophagus While trauma from intubation prior to death was apparently a factor in about one third of the cases, the relationship was not clear, since the pathologic changes in the esophagus were the same as those in which no intubation had occurred

3 The fact that esophagitis as well as ulcer of the esophagus occurs almost invariably in the distal segment lends further support to this etiologic relationship, since this is the anatomic area which would be bathed more intimately by the regurgitation of acid gastric secretion

Nausea and vomiting may force the acid gastric secretion into the distal esophagus, and it is conceivable that the repetition of such action over a long period of time may irritate the mucosa of the esophagus and eventually lead to inflammatory changes with either stricture or ulceration which may go on to perforation The deleterious effect of severe vomiting on the integrity of the esophagus is well recognized That it may even be a cause of actual rupture of the esophagus was shown many years ago in the classical account by Dryden⁶

Sir J—P—, of his Majesty's 10th regiment of foot, was seized on the morning of the 1st of December 1787 (after taking a little breakfast), with nausea, and inclination to vomit, which he encouraged by drinking plentifully of warm water, until it produced full vomiting He had an idea, which is very common in this country, that his stomach was loaded with bile, and, of course, its evacuation necessary to his future health However, in this he certainly was mistaken, for the irritability of the stomach proceeded from a debauch over-night, with which he had been much inebriated

The warm water caused him to strain very much, and in this action he felt something give way internally, which gave him a sensation as if he had received an injection of some liquid matter into the cavity of the thorax He also brought up a little blood, from whence he conjectured, that one of the large blood-vessels of the lungs was ruptured, and occasioned the acute pain he then began to feel about the region of the stomach and abdomen

APPEARANCES ON DISSECTION

On perforating the cavity of the thorax, a considerable quantity of air escaped The cellular substance was found much distended with air, particularly about the neck The oesophagus was found ruptured, before it passes into the diaphragm, and the whole contents of the stomach had passed through that orifice into the cavity of the thorax, and compressed the lungs into a very small compass About one gallon, consisting of a mixture of wine, water, and the matter employed as aliment, was taken out of the left side, where he felt all his pain Nearly two quarts of the same were also found effused in the right side, and in an effervescent state

The laceration in the oesophagus was longitudinal, and so large as to admit the fore and middle finger

The potential relationship of vomiting to the development of ulceration of the esophagus may be found in the interesting description by Dr Golding Bird⁷ under the heading of "The Pathology of Morbid Digestion" in the *London Medical Gazette*, 1841, volume 29, page 383

I saw, in consultation with my friend Dr Hopkins, Miss S—— aet 29, a lady highly educated and of remarkably cultivated taste she had been under the care of Dr Hopkins occasionally for the last three years, for pyrosis, remarkable chiefly for the very large quantity of fluid ejected from the stomach To this she had been subject, more or less, and at irregular intervals, during the last six years, at which period the affection insidiously appeared In general, the vomiting yielded to the remedial measures adopted For some weeks prior to my seeing her, this lady had laboured under almost constant pyrosis, complete loss of appetite, amounting to horror at the sight of food, and rapid emaciation The latter was partly accounted for by the existence of tubercular disease in both lungs she had lost two sisters with phthisis during the last two years

Autopsy On removing the stomach with the oesophagus with pharynx, and slitting them up from the cardiac extremity, the whole tube was found denuded of epithelium to within half an inch of the stomach, where excessive congestion and some spots of ecchymosis were met with The muscular coat of the oesophagus was very considerably hypertrophied The whole length of the tube

was studded with ulcers varying in size from a split pea to a four penny piece, and penetrating to the muscular coat some of these ulcers extended into the pharynx its mucous lining was, however, exceedingly injected. A preparation has been made of the oesophagus, which is placed in the Museum of Guy's Hospital.

In this very interesting case, a question arises as to the necessary connection between the ulceration of the oesophagus and the pyrosis, and how far the latter depended upon, or was produced by, the peculiar position of the stomach. I have met with two other cases of oesophageal disease, attended with pyrosis in one of the ulceration, and in the other stricture existed.

4 Further support of the acid gastric theory may be found in the apparent effectiveness of subtotal gastrectomy as reported by Wangensteen and Leven.⁸ The purpose of the resection was to produce a reduction of the gastric acidity and of the digestive capacity of the secretion of the remaining portion of the stomach. There was also an accelerated emptying of the gastric contents as a result of this surgical procedure. Both these factors diminished the possibility that regurgitated acid secretion was perpetuating the esophagitis. They claimed that this procedure was effective in causing a regression of the esophageal stricture.

ROENTGEN DIAGNOSIS

In the deformity produced by esophagitis the area of involvement is apt to be narrowed. The contour is smooth. There is no sharp demarcation of the narrowed area of the esophagus from the rest of the smoothly outlined contour, the one passing imperceptibly into the other.

In the narrowing produced by a new growth, however, the area of involvement is irregular, and there is sharp demarcation of this region from the rest of the esophagus. In esophagitis, while the mucosal appearance of the involved area is altered with increased prominence of the folds and occasionally the presence of translucent areas because of superficial excrescences, these deformities, as a rule never assume the

marked irregularities produced by a new growth, invading the wall or extending into the lumen in polypoid fashion.

In a borderline case, esophagoscopy and biopsy may be essential in order to arrive at a differential diagnosis.

Illustrative Cases The following reports illustrate cases of peptic esophagitis.

J. G., male, aged 58. For 8 months this patient noticed that meats when improperly chewed, stuck in his throat before going down. He had no pain or evidence of bleeding. There was no increased difficulty in swallowing. Two years previously the patient had vomited blood and remained in the hospital for a week. He had lost about 12 pounds.

Esophagoscopy revealed a stricture of the esophagus, at the level shown in the roentgenogram, at the lower extremity of which there was ulceration of the mucosa. The presence of ulceration was noted on repeated examinations. At no time did esophagoscopy reveal any evidence of new growth.

A biopsy of tissue removed at the site of stricture was reported as 'chronic productive inflammation.' The patient gradually improved as a result of dilatation of the strictured area of the esophagus.



FIG. 92 Smooth narrowing of the esophagus due to esophagitis.

Roentgen examination (Fig 92) revealed a very smoothly outlined area of stenosis at the junction of the lower and the middle thirds of the esophagus, with moderate dilatation above this region. Because of the smooth, sharply outlined appearance of the area of constriction of the esophagus in the roentgenogram, the strictured area was considered to be the result of a benign process. In conjunction with the findings on esophagoscopy examination, as well as the pathologic findings on biopsy, this may be considered a case of peptic esophagitis.

The next case has features of unusual interest.

L. W., male, aged 63. Six months before his admission to the hospital, this patient complained of a sudden onset of vomiting. On three occasions he brought up blood. He complained of difficulty in swallowing solid foods, but he had no pain as a result of this difficulty in swallowing. Gastrostomy was performed.

The esophagoscopy examination revealed a firm, fibrous, cicatrizing stenosis 3.5 cm from the upper teeth. This stenosed area was dilated through the esophagoscope.

A specimen was removed for biopsy. It proved to be scar tissue. The patient's difficulty in swallowing disappeared, and the

gastrostomy opening was closed. Apparently the stricture of the esophagus had resulted from previous ulceration.

Roentgen examination of the esophagus prior to esophageal dilatation (Fig 93 A) showed an area of marked stenosis about 1 inch long. Above it the esophagus showed considerable dilatation. Following mechanical dilatation of the involved region, the strictured area showed a considerable increase in lumen (Fig 93 B).

This patient had been followed for a period of several years, and there was no return of the original stricture. Repeated roentgenographic studies showed an appearance similar to that seen in Figure 93 B. In all probability, this stenosis arose as the result of ulceration, with secondary constriction. The history of the vomiting of blood at the onset of the disorder is clinically suggestive of active ulceration of the esophagus at that time.

A well-controlled example of esophagitis is illustrated by the following case.

F. H., male, aged 58. During the preceding 3 years the patient had had increasing difficulty in swallowing food, especially solids, with a feeling that the food was sticking in his chest. This was associated with substernal pain radiating to the back. He vomited oc-

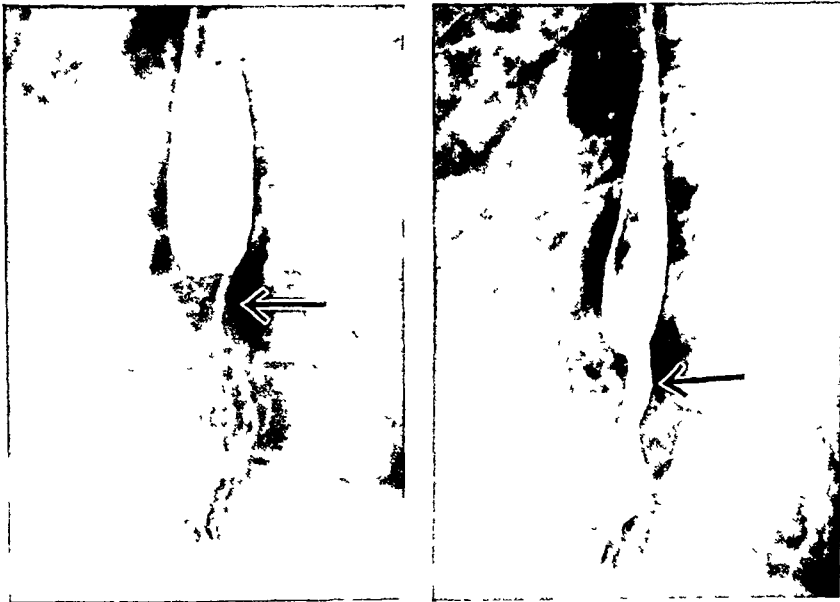


FIG 93 (A, Left) Marked narrowing of the esophagus due to benign stenosis. (B, Right) Same patient. Note the marked improvement after dilatation of the stenosed area.

asionally and sometimes bright blood was present. He had lost 20 pounds in the preceding 3 months. Physical examination revealed a pale, weak male. There was tenderness in the epigastrium, but examination of the abdomen was otherwise essentially negative.

Esophagoscopy showed an ulceration of the mucosa just above the cardia. A biopsy of this region was taken. The pathologic report was acute and chronic esophagitis.

A biopsy of the involved area of the esophagus was taken again 3 weeks later, and pathologic examination at this time was reported as hyperplasia of the esophageal epithelium. There was no tumor tissue. A third biopsy 5 weeks later was reported as showing an acute inflammatory exudate.

The patient was followed both in the outpatient department and on a number of occasions on the ward. Repeated esophagoscopy examinations were made, all of which were negative for new growth.

Examination 3 years after the original esophagoscopy showed a smooth stricture at the distal end. There was no evidence of new growth. The stricture was dilated, with considerable clinical improvement.

Roentgen examination (Fig. 94) showed a comparatively smooth narrowing at the junction of the lower and the middle thirds, interpreted as indicating a stricture of inflammatory origin which might be associated with ulceration even though a niche was not actually demonstrable. The roentgenographic appearance represented the condition 3½ years after the original observation at which time a similar diagnosis had been made. The diagnosis of benign stricture thus received further support from the duration of the deformity (3½ years) without any evidence that irregularity had developed in the narrowed zone. The diagnosis was further substantiated by repeated esophagoscopy examinations and pathologic studies of biopsies taken on numerous occasions. Also the

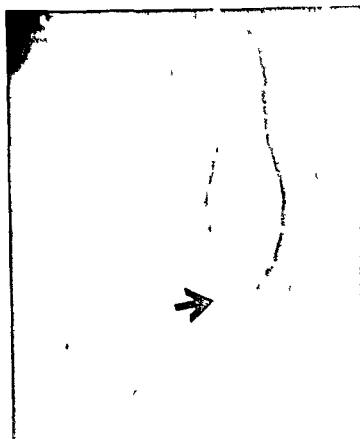


FIG. 94 Peptic esophagitis. Note the smooth narrowing of the esophagus.



FIG 95 Peptic esophagitis. Note the smooth narrowing of the esophagus proximal to the cardiac end. This differentiates the condition from cardiospasm, in which the tapering occurs at the cardiac end.

patient benefited clinically from dilatation of the stricture under esophagoscopy control. Incidentally, a duodenal ulcer was found originally at the time when the esophageal stricture was first discovered. The patient was later operated on for this, and a partial gastrectomy was done. The possibility arises that the duodenal ulcer may have antedated the esophageal lesion and that repeated vomiting of hyperacid fluid might have been an etiologic factor in the development of the esophageal inflammation.

Because of recurrent episodes of dysphagia and hemorrhage, operation was performed with resection of the distal 10 cm of the esophagus and an anastomosis of the remaining esophagus to the stomach. The pathologic diagnosis of the resected segment was chronic esophagitis.

The findings in the next case were confirmed by autopsy.

J. G., male, aged 59, gave a history of dysphagia of 2 months' duration and a weight loss of 20 pounds. He vomited undigested food, which did not contain blood. At first, the dysphagia was limited to solid food but later he vomited liquids as well. Ingestion of food was accompanied by pain, which was relieved by vomiting. Physical examination was essentially negative except for tenderness in the epigastrium.

Autopsy revealed an intense esophagitis. The inflammatory tissue consisted mainly of plasma cells, and infiltrated the nerve ganglia and muscle fibers.

Roentgen examination (Fig 95) revealed a smooth narrowing of the esophagus proximal to the cardiac end, with secondary dilatation above. The extreme distal portion of the esophagus was not involved, thus differentiating it from the tapering of cardiospasm. The smooth narrowing was characteristic of a benign inflammatory process as was verified at autopsy.

Almost complete occlusion of the distal portion of the esophagus as the result of a benign esophagitis is illustrated by the following case.

R. C., male, aged 62. Four years before admission to the hospital the patient complained of food sticking behind the sternum. This was relieved by vomiting. These symptoms recurred intermittently, and then he developed a progressive dysphagia so that 5 days before admission he had great difficulty in swallowing even liquids. Occasionally, the vomitus was blood-tinged. The substernal pain became worse. He lost 40 pounds in the preceding 4 years. Physical examination was essentially negative. Repeated esophagoscopy examinations showed evidence of esophagitis with stricture. Microscopic examination of a biopsy of the lesion showed chronic inflammatory pathology. Because of the persistent dysphagia the strictured portion of the esophagus was resected, and an esophagogastrostomy was done. Macroscopic examination of the resected esophagus "there is a stricture through which only a 1-mm probe can be passed. The mucosa here appears intact and is surrounded by a thick firm yellowish band 1 cm long and 1 cm thick and extending to the serosa."

Microscopic Examination "One section through the junction of esophagus and stom-

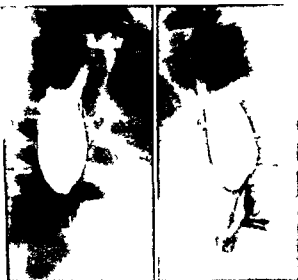


FIG 96 Esophagitis Note the narrowed distal portion of the esophagus. It is of essentially smooth contour (A Left) Right oblique position (B Right) Left oblique position

ach reveals marked fibrosis underlying the stratified squamous cell epithelium of the esophageal mucosa. The process extends in places deep into the muscularis and there is some serosal fibrosis as well.

Diagnosis Chronic esophagitis with fibrosis and stricture formation.

Radiographic examination (Figs. 96 A and B) shows the narrowed segment of the esophagus due to benign esophagitis.

An irregularity of the contour of the esophagus due to benign stricture but simulating that of malignant infiltration may be noted in the next case.

J C male, aged 26. Eleven years previously the patient had a gastrostomy done because of a stricture of the esophagus which had been characterized clinically by a 2 month history of progressive dysphagia so that finally he was able to swallow only fluids. The stricture was treated repeatedly by instrumental dilatation. He did very well, was able to swallow without difficulty and gained 40 pounds in weight. Finally the gastrostomy was closed. Later he returned complaining of abdominal pain. Radiographic examination revealed the presence of an ulcer of the first portion of the duodenum. The dysphagia had not recurred.

Incidental radiographic examination of the esophagus at that time 11 years after the

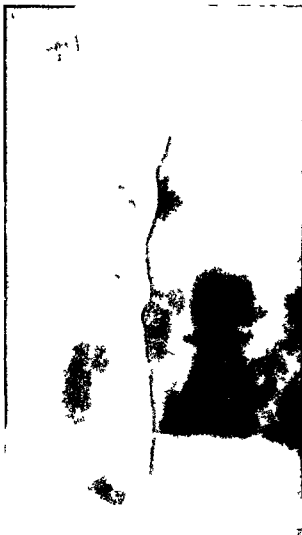


FIG 97 Esophagitis Note the irregular configuration of the narrowed involved portion of the esophagus simulating that of new growth. The deformity is apparently due to two factors (1) the underlying esophagitis, (2) the reaction to the trauma of repeated esophagoscopy examinations and instrumental dilatations.

onset of his dysphagia, showed an irregular narrowing in the distal portion of the esophagus with moderate dilatation proximal to the area of involvement (Fig. 97). On the basis of the radiographic evidence alone I do not think it would be possible to state with assurance that the lesion was benign. However, the repeated esophagoscopy studies, the successful clinical response with the gain of 40 pounds in weight, the failure of recurrence of symptoms referable to the esophagus during a period of 11 years all obviously pointed to the benign nature of the lesion. The deformity



Figs 101 and 102 Stenosis of the esophagus due to lye

The following is a case of this nature

G P About 4 years before his admission to the hospital, this patient had swallowed an indeterminate quantity of lye. A Levin tube was left *in situ* thereafter for 4 weeks. The patient had frequent esophageal dilatations for 1½ years. In the following year, he had no treatment because he was relatively symptom free. Three weeks before admission to the hospital his difficulty in swallowing returned and became progressively worse until he could barely swallow water.

The esophagoscope was passed, and a stricture was noted with only a small pin point opening. The stricture opening expanded and closed with inspiration and expiration. Repeated dilatations of the esophagus were done. The lumen gradually increased in size and the patient was discharged improved.

Roentgen examination (Fig 99) revealed a marked stenosis of smooth outline involving the midportion of the esophagus, with moderate dilatation of the esophagus above the area of constriction. There was no evidence of any irregularity of contour anywhere throughout the esophagus. The very sharply defined contour at the site of narrowing strongly favored the diagnosis of a benign constricting process. The exact cause in this case could be determined only on the basis of the clinical history and the evidence on esophagoscopy examination.

Figure 100 is an example of diffuse narrowing of almost the entire esophagus, with irregularity of contour due to the swallowing of lye.

Additional examples of stricture of the esophagus resulting from caustics may be noted in Figures 101 and 102.

In the next case of stricture of the esophagus resulting from lye, a fistulous tract ultimately developed.

M D female aged 18. The patient had tried to commit suicide by swallowing a fifth of a glass of a combination of Lysol and lye. She vomited blood streaked material and was promptly removed to the hospital. The following day she complained of a substernal burning pain and dysphagia. A Levin tube was passed through the esophagus for feeding. This was removed about 2 weeks later, following which she was able to eat the regular ward diet.



FIG 103 Stenosis of the esophagus due to lye, with fistula

Esophagoscopy at this time revealed a stricture without any hemorrhagic areas.

After the patient was discharged from the hospital the stricture was treated by weekly dilatation of the esophagus. In spite of this the dysphagia increased to such a degree that a Levin tube could not be passed. A gastrotomy was done.

Roentgen examination 9 months after the swallowing of lye (Fig 103) showed marked stricture of the esophagus with an irregularly outlined, walled off, fistulous tract originating at the junction of the upper and the middle thirds of the esophagus. What had evidently occurred was a perforation of the esophageal



FIG 104 Esophagitis In addition to the constriction of the esophagus note the marked abnormality of the mucosal pattern as indicated by the numerous rounded translucent areas of varying sizes indicative of polypoid changes of the mucosa

wall, possibly induced at the time of one of the dilatations of the stricture through the esophagoscope

The patient continued to have increasing

difficulty in swallowing as well as episodes of bleeding, necessitating further surgical intervention. At operation the esophagus below the arch of the aorta was found to be fibrous and scarred so that it was impossible to identify its muscle structure. The involved portion of the esophagus was resected, and an esophagogastrostomy was done.

Pathologic examination revealed that a segment of the lower esophagus had been opened longitudinally. The wall was definitely thickened throughout, and the lumen appeared to be markedly narrowed at the lower end.

Microscopic examination revealed (1) chronic esophagitis, (2) fibrosis of esophagus with hypertrophy of muscle, (3) foreign-body giant cell reaction.

The abnormal changes of the mucosal pattern of the esophagus produced by the ingestion of lye may be noted in Figure 104. The longitudinal folds have been replaced by rounded translucent areas of varying size.

The patient, S. H., male, 18, had a solution of lye thrown in his face, striking both eyes as well as entering his throat. Complete searing of both eyes occurred as well as a stricture of the esophagus. Esophagoscopy showed a constriction of the esophagus about 27 cm from the teeth. A No. 18 bougie was passed. A No. 20 bougie could not be passed. An unsuccessful attempt was made to pass a feeding tube into the stomach. In spite of the constriction the patient was able to swallow liquids and soft foods. Attempts were made to dilate the stricture under esophagoscopy control. The persistent constriction of the esophagus required a gastrostomy. Later a partial esophagectomy and an esophagogastrostomy were done above the arch of the aorta.

The progressive constriction of the esophagus following the administration of lye is shown in the following case.

J. B., male, aged 39. This patient swallowed lye in an attempt at suicide. Figure 105 A shows the appearance 9 days after the swallowing of the lye. Figure 105 B shows the appearance 10 days later. Note the increase in the degree of constriction, as well as the irregularity of its contour due in part to actual ulcerations. The extreme distal portion of the esophagus is completely constricted. The progressive character of the obstruction may be noted by the radiographic appearance in Figure 105 C. This examination was made



FIG 10s (A, *Top left*) Stricture of the esophagus shortly after the ingestion of lye (B, *Top right*) Appearance 10 days later. The constriction is now more marked, and irregularity of contour has supervened (C, *Bottom left*) Appearance 8 months later. Note the complete occlusion of the distal portion of the esophagus at this time (D, *Bottom right*) Appearance following resection of the esophagus and esophagogastrostomy

8 months later. The persistent occlusion of the esophagus necessitated a resection of most of the distal portion of the esophagus and an esophagogastrostomy. Pathologic examination of the resected esophagus revealed chronic esophagitis and periesophagitis with fibrosis and marked cicatricial stenosis. Figure 105 D

shows the radiographic appearance postoperatively. Note the unusually high position of the anastomosed stomach.

The association of esophagitis and esophageal ulcer with the short esophagus will be discussed in Chapter 16.

REFERENCES

- 1 Jackson, C. Peptic ulcer of the esophagus. *J A M A* 92 369, 1929.
- 2 Vinson, P. P. Cicatricial (benign) stricture of the esophagus of unknown origin: report based on forty cases. *Surg, Gynec & Obst* 52 955, 1931.
- 3 Jackson, C. L. Esophageal stenosis associated with ulcer of the stomach and duodenum. *Tr Am Therap Soc*, p 143, 1932.
- 4 Hertzog, A. J., and Leighton, R. Spontaneous perforation of esophagus. *Minnesota Med* 29 442, 1946.
- 5 Bartels, E. C. Acute ulcerative esophagitis, a pathologic and clinical study of 82 cases observed at necropsy. *Arch Path* 20 369, 1935.
- 6 Dryden, John. An account of a rupture of the oesophagus from the action of vomiting. *Medical Commentaries* 3 308, 1788.
- 7 Bird, Golding. The pathology of morbid digestion. *London Med Gaz* 29 383, 1841.
- 8 Wangenstein, O. H. and Leven, N. L. Gastric resection for esophagitis and stricture of acid-peptic origin. *Surg, Gynec & Obst* 88 560, 1949.

The Esophagus in Scleroderma and the Collagen Diseases

Scleroderma is a systemic disease involving a disorder primarily of the connective tissue. The diffuse involvement of the collagen system has also been noted in lupus erythematosus, dermatomyositis and periarteritis nodo. The alteration of the skin is only one manifestation of this generalized disorder. The increased connective tissue proliferation produces an interstitial myositis with secondary atrophy. The vascular structures may partake in the disease with localized arterial obliteration. The lesions of scleroderma may be found in most of the internal viscera as well as the bones and the joints and the bone marrow. Lesions of the ears and the eyes may develop also.

Therefore the changes in the alimentary tract represent localized manifestations of this widespread disease.

The first recorded cases of the association of dysphagia and scleroderma were those of Ehrmann,¹ Schmidt-Helm² and Schwarz.³ None of these authors, however, had the opportunity of studying the underlying pathology of the esophagus responsible for the dysphagia, although they suspected that the changes were part of the more generalized disease and similar to those in the skin.

The proliferation of connective tissue in the wall of the esophagus to explain the derangement of function was described at autopsy by Matsui, quoted by Kure,⁴ Hoelsi⁵ and Weissenbach.⁶ On histologic examination of the esophagus in their cases of scleroderma these investigators found in duration of the submucosa, degeneration of elastic tissue and hypertrophy of the

muscularis mucosae, as well as atrophy of the muscular layer, with vascularization and increased proliferation of connective tissue. The presence of serious alterations in the structural integrity of the wall of the esophagus was further corroborated by the autopsy studies of Rake,⁷ Weiss,¹⁰ Bevans¹¹ and Goetz.¹² As a result of these changes in the wall of the esophagus, there are a number of disturbances in its behavior, which may be recognized on fluoroscopic and roentgen examination.

1. There is a marked diminution or complete absence of peristaltic activity, the lumen of the esophagus remaining wide open even though there is no stenosis.

2. There is considerable delay in the emptying of the esophagus in the absence of constriction, and the barium may cling to the wall for many hours. Detailed studies of the disturbed function of the esophagus in scleroderma were recorded by Kure and his associates⁴ in five cases.

3. In some cases there may be an actual area of stenosis at the distal end of the esophagus. Fessler and Pohl¹³ in one of their roentgenograms showed an area of constriction about 2 cm in length and of smooth contour, with secondary dilatation of the proximal portion. This region was visualized by esophagoscopy, but no tissue was removed for biopsy. They considered the narrowing to be secondary to connective tissue proliferation with scarring. Their radiographic findings were confirmed by other observers (Ochsner and DeBailey,¹⁴ Jackson,¹ Weiss,¹⁰ Lindsay,

Templeton and Rothman,¹⁶ Hale and Schatzki,¹⁷ Dorken¹⁸ and Olsen, O'Leary and Kirklin¹⁹)

At times a considerable portion of the distal esophagus may be constricted. A smooth stricture of the distal third of the esophagus was present in the case of progressive scleroderma reported by Schwarz and Skinsnes.²⁰ The patient also showed a destructive lesion of the terminal phalanges. At autopsy, examination of the esophagus revealed mucosal erosions, submucosal fibrosis and hypertrophy of the muscularis mucosae. Very significant was the fibrous atrophy of the inner layer of the tunica muscularis. In Goetz's case¹² autopsy revealed an oval chronic ulcer about 1 inch above the cardiac end. The wall of the esophagus at the site of the ulcer and in the adjacent area was markedly thickened. There were also numerous small elevated white patches of leukoplakia in the distal half of the esophagus.

Lindsay and his associates¹⁶ carried out biopsy studies of tissue removed through the esophagoscope from the area of stenosis, and microscopic examination showed changes in the connective tissue characteristic of the alterations produced by scleroderma in the skin. As a result of the stricture, the esophagus may be shortened and in the process may pull up the cardiac portion of the stomach into the chest through the hiatus (Olsen, O'Leary and Kirklin).¹⁹ The appearance may mimic that of a congenitally short esophagus.

4 In some cases disturbance in the behavior of the esophagus in scleroderma may be present in the absence of any subjective symptoms (Kur  ,⁶ Bevans,¹¹)

Presumably, therefore, the organic involvement of the esophagus must be quite advanced before the patient complains of dysphagia.

Illustrative Case Abnormalities of the esophagus in scleroderma are illustrated by the following case.

M. W., female, aged 53. During the preceding 4 years, the patient noted that the

skin over her hands became tense and appeared white over the knuckles. The fingers became contracted, with difficulty in extension, which gradually became more marked. During this period of 4 years, the patient had difficulty in swallowing, greater with solid food but also some with liquids. The food seemed to stick substernally for about 2 hours. Occasionally, drinking water helped to push the food down. Her dysphagia was lessened if she minced her food very fine and ate very slowly. She had lost 20 pounds in the preceding 3 months.

Physical examination revealed that some of the fingers were permanently flexed at the terminal phalanges. A similar condition existed in the lower extremities and the toes. The skin over the hands was atrophic, dry and tense. The terminal phalanges of the second, third, fourth and fifth fingers of both hands were held in a flexed position, increasing in flexion in that order. There was some depigmentation of the skin over the heads of the metacarpals. The nails showed changes, with subungual keratosis of the index fingers. There was some pitting and striation of the nails. Similar changes were present in the toes. There was wrinkling, dryness and slight contraction of the skin about the lips.

Roentgen examination revealed hypertrophic osteoarthritis of both wrist joints. There was considerable osteoporosis, involving the bones of the hands and the wrists. There was absorption of the distal portion of the terminal phalanges of both thumbs, more extensive on the left side, but to some extent affecting the distal phalanges of the other fingers.

Examination of the esophagus (Fig 106 A) showed moderate dilatation throughout. Very little of the barium entered the stomach during prolonged fluoroscopy (about 15 minutes) and in spite of repeated efforts at swallowing on the part of the patient. One half hour later (Fig 106 B) much of the substance was still clinging to the wall. At this time, there was a blunt, abrupt termination of the distal end. At the end of 13 hours, fluoroscopic observation still showed a small amount of barium in the esophagus. The contour of the wall of the esophagus appeared quite smooth. At times, on examination in the second or left oblique position (Fig 106 C), there appeared to be some constriction of the extreme distal end. The walls of the esophagus at no time showed evidence of contraction but remained as if rigid. Emptying appeared to occur primarily as the result of gravity and the

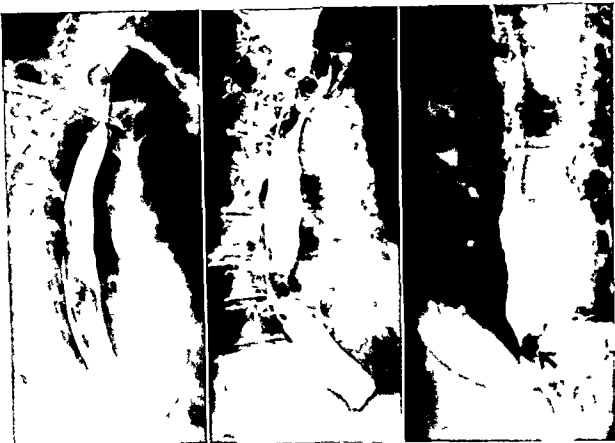


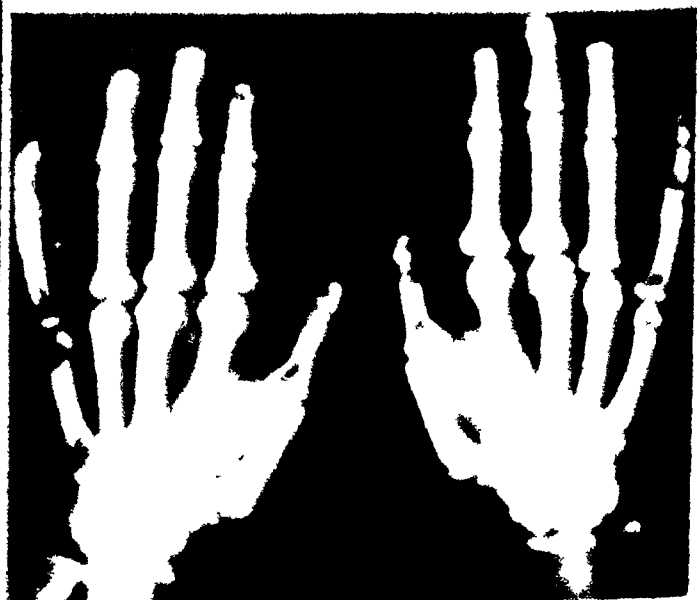
FIG 106 (A *Top, left*) The esophagus in scleroderma. Very little of the barium entered the stomach during prolonged fluoroscopy in spite of repeated efforts at swallowing on the part of the patient. No peristaltic activity was noted (B, *Top, center*) Same patient. Appearance one half hour after the swallowing of barium. Note the marked delay in emptying (C, *Top, right*) Same patient. Constriction at the distal end of the esophagus seen in the second oblique position (D, *Bottom*) Kymograph of the esophagus in this case



occasional opening up of the distal cardiac end particularly under the influence of repeated acts of deglutition. Figure 106 D is a kymograph of the esophagus in this case.

The dilatation and atony of the esophagus in scleroderma may simulate the appearance in achalasia of the cardia. Figure 107 A is the radiographic appearance of the esophagus. Note the abnormal dilatation, particularly of the distal half. Figure 107 B shows the destruction of the terminal phalanges of the fingers of both hands.

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skin over the ribs was thickened, scaly, dry, with some areas of excoriation. The distribution was symmetrical, involving the extensor surfaces of both arms, the backs of the fingers and the hands, the face across the forehead and the cheeks, the anterolateral and posterior portions of the neck, the chest above the breasts, the back from the sixth to the eighth dorsal vertebra to the costovertebral angle, the lumbar areas as well as the knee caps and the suprapatellar areas. The tips of the fingers showed no necrosis. There were no joint disorders. There was evidence of muscular weakness, and it was the clinical impression that she had a muscular dystrophy secondary to dermatomyositis. Electromyographic studies were done on the right deltoid and the right sternocleidomastoid muscle. The muscles

were clinically weak and atrophic. The findings were consistent with myogenic atrophy. The skin lesions improved under ACTH therapy with little if any improvement in muscular function. A biopsy of the skin and the muscle of the left deltoid was studied microscopically.

Muscle section reveals areas of atrophy of muscle fibers and an occasional necrotic muscle fiber. In the areas of most marked muscle change and also about blood vessels there is a mild to marked infiltration of lymphocytes, large mononuclear cells, occasional eosinophiles and neutrophils. A rare multinucleated giant cell is present. Also in the areas of muscle degeneration and atrophy there is proliferation of the cells of the sarcolemma sheath. The dermis shows an increase



FIG. 108. Appearance of the esophagus in scleroderma. (A, Left) Promptly after the ingestion of barium suspension. (B, Right) Examination 45 minutes later. Note that at this time a small amount of barium still clings to the wall of the esophagus (no history of dysphagia).



FIG 109 The esophagus in a case of dermatomyositis. Fluoroscopically, it appeared to be atonic, and the barium remained within it for many minutes in spite of the fact that the patient repeatedly went through swallowing motions. Note also the area of constriction near its distal end. The appearance is similar to that seen in some cases in which the underlying pathology is scleroderma.

of collagen within the superficial portion of the corium, and a mild perivascular chronic inflammatory reaction.

"Diagnosis Chronic dermatitis, chronic myositis

"Note The changes are consistent with the diagnosis of dermatomyositis."

The radiographic findings in the examination of the esophagus are quite interesting in view of the nature of the underlying disease (Fig 109). In spite of the clinical absence of dysphagia, it was observed that the esophagus appeared to be atonic. The patient had considerable difficulty in swallowing the barium. Although the patient went through the motions of swallowing, there was very little effect upon the emptying of the esophagus so that the barium remained within it for an abnormally long period of time. In addition there was an area of constriction at the distal portion of the esophagus just proximal to the extreme cardiac end. Fluoroscopically, there was also a delay in the passage of the barium from the hypopharynx into the esophagus. The appearance and the behavior of the esophagus were reminiscent of those seen in scleroderma. It is conceivable that the pathologic changes of the underlying dermatomyositis may have involved the wall of the esophagus, thus being responsible for its functional aberrations of behavior as well as the moderate degree of stricture at its distal end.

An additional example of the roentgen appearance of the esophagus in scleroderma may be noted in Figure 452 A, which is included later in this book in connection with changes which had occurred in the small intestine.

REFERENCES

- 1 Ehrmann, S. Über die Beziehung der Sklerodermie zu den autotoxischen Erythemen, *Wien med Wochenschr* 53 1097, 1156, 1903.
- 2 Schmidt, R. Sklerodermie mit Dysphagie, *Med. Klin* 12 401, 1916.
- 3 Helm, F. Seltene Röntgenbilder des Oesophagus, *Med. Klin* 14 665, 1918.
- 4 Schwarz, Paul. Sklerodermie und Röntgenkastration, *Schweiz med Wehnschr* 56 246, 1926.
- 5 Matsui, S. Über die Pathologie und Pathogenese von Scleroderma universalis, *Mitt med Fakultät Tokyo* 31 55, 1924.

- 6 Kure, K., Tamagata, K., Isukada, S., and Hivoshi, J. Passagestörung des Oesophagus bei Sklerodermie und Dysphagia musculorum progressiva, *Klin Wchnschr* 15 516 1936
- 7 Hoesli, H. Troubles fonctionnels et lésions de l'oesophage dans la sclerodermie theses Paris 1937
- 8 Weissenbach, R. J., Martinero Bouwens Pizon, and di Matteo. Sclerodermie progressive Bull Soc franc dermat et syph 44 2018, 1937, and Weissenbach, R. J., Stewart, W. M., and Hoesli, H. Troubles fonctionnels dans la sclerodermie, *Ann dermat et syph*, series 7, 9 81, 193 1938
- 9 Rake, S. G. On the pathology and pathogenesis of scleroderma Bull Johns Hopkins Hosp 42 212 1931
- 10 Weiss, S. Stead, I. A. Jr Warren, J. A., and Bailey, O. T. Scleroderma—heart disease with consideration of certain other visceral manifestations of scleroderma, *Arch Int Med* 71 749 1943
- 11 Bevans, M. Pathology of scleroderma with special reference to the changes in the gastrointestinal tract Am J Path 21 25 1945
- 12 Goetz, R. H. The pathology of progressive systemic sclerosis (generalized scleroderma) with special reference to changes in the viscera J Capetown Postgrad M A 4 337 1945
- 13 Lessler, A. and Pohl, R. Stenosierender Prozess des Oesophagus bei Sklerodermie Dermat Zeitschr 63 164, 1932
- 14 Ochsner, A., and DeBakey, M. Scleroderma, surgical considerations, New Orleans M & S J 92 24, 1939
- 15 Jackson, James Roentgen features of scleroderma and acrosclerosis, *Radiology* 40 163, 1943
- 16 Lindsay, J. R., Templeton, F. L. and Rothman, S. Lesions of the esophagus in generalized progressive scleroderma J A M A 123 745 1943
- 17 Hale, C. H. and Schitzki, R. The roentgenological appearance of the gastrointestinal tract in scleroderma, *Am J Roentgenol* 51 407 1944
- 18 Van Dorken, H. Über Sklerodermie mit Oesophagus Beteiligung, *Radiologica Clinica* 20 129, 1951
- 19 Olsen, Arthur M. O'Leary, Paul A., and Kirklin, B. R. Esophageal lesions associated with acrosclerosis and scleroderma Arch Int Med 76 189 1945
- 20 Schwarz, G. S. and Skinsnes, O. K. Generalized progressive scleroderma, *Am J Roentgenol* 62 359, 1949

Syphilis and Tuberculosis of the Esophagus

SYPHILIS

Incidence Cornil¹ stated that syphilis rarely attacks the esophagus. He quoted a case reported by Virchow in which a gumma was found at autopsy, but he had encountered no such case in his own experience.

Lublinski² gave an excellent review of the older literature on syphilis of the esophagus and described two cases of his own, these, however, were not corroborated by autopsy study. The stricture in both these cases diminished following antisyphilitic treatment. Both patients had given a history of syphilis, and in one case the esophageal lesion was associated with a gumma of the tongue.

Wilks³ described a case of typical syphilis of the pharynx in which the ulcerations extended to the esophagus with resulting contraction of the esophagus.

Wile⁴ described a case of syphilis of the esophagus in a woman, aged 40, with a history of dysphagia of about 9 years' duration, which had gradually increased in severity. Esophagoscopy showed a marked sclerosis of the entire tube, so that only the smallest-sized filiform bougie could be passed through it into the stomach. Roentgen examination showed two areas of stricture. Excellent clinical response was obtained after antisyphilitic measures. However, dilatation of the stricture was also carried out with bougies of increasing size.

Guyot⁵ collected 57 cases of syphilis of the esophagus reported in the literature, to which he added 2 cases of his own. Of these he stated that 11 cases were verified by examination at autopsy, and 15, diagnosed

esophagoscopically, were cured by antisyphilitic measures. He described three anatomic types: (1) the presence of a gumma, which may undergo ulceration, (2) the spread of a syphilitic phlebitis or specific pneumonitis, with extension to the esophagus or secondary perforation into it, and (3) a diffuse chronic inflammation, with generalized sclerosis and superficial ulceration. All three forms may terminate in stenosis.

Tertiary syphilis of the esophagus has been described by Wilcox.⁶ In a case which he examined roentgenologically, an irregularity of contour involving almost the entire esophagus, giving the appearance of a diffuse esophagitis, was seen. Improvement followed dilatation and antisyphilitic measures, but no anatomic diagnosis was made.

Because of the rarity of the condition, possibly only those cases should be accepted which meet the criteria of anatomic and bacteriologic proof.

Roentgen Characteristics Roentgenologically, no specific characteristics have been noted, and the appearance has simulated that either of a nonspecific inflammatory process or a new growth.

TUBERCULOSIS OF THE ESOPHAGUS

Tuberculosis of the esophagus is extremely rare. Rokitansky⁷ in his famous work stated that tuberculosis of the esophagus is rarely if ever found, and he warned that this must not be confused with "tubercular degenera-

tion of the neighbouring lymphatic glands.' He apparently had had no personal experience with a case of intrinsic tuberculosis of the esophagus.

Flexner⁸ described three types of tuberculous involvement of the esophagus. In the first type, the esophagus is secondarily involved through contiguity, as when caseous bronchial nodes ulcerate through into the esophagus or when an abscess associated with caries of the vertebrae perforates into it. Under this heading he also included secondary extension to the esophagus from tuberculous ulcers of the pharynx.

In Group 2 he included those cases in which the existence of a previous lesion predisposed the mucosa to tuberculous infection.

To the third group belong those cases in which involvement of the esophagus occurred in the course of a general infection of the body in acute, disseminated milary tuberculosis.

Flexner in his review of the subject of tuberculosis of the esophagus up to the time of the publication of his paper in 1893 stated that Spillman⁹ in 1878 described the first case of an intrinsic lesion of tuberculous nature which had developed within the esophagus itself. An ulcer was present 1 cm by 1.5 cm in the midportion of the esophagus, with a barely perceptible perforation into the left bronchus. The patient had a pleural tuberculosis.

The only other cases that met the criteria (according to Flexner) of Group 3 were those reported by Frerichs¹⁰ in 1882 and Mazzotti¹¹ in 1885. To these reports Flexner in his article added a case from his personal experience.

Denonvilliers¹ is frequently given credit for having reported in 1837 the first case of tuberculosis of the esophagus. In his description, however, it is to be noted that he considered the ulceration of the esophagus which had produced a fistulous communication with the trachea as being secondary to a tuberculous process in the intercellular space between the trachea and the esoph-

agus. Therefore, this case cannot be included in the third group described by Flexner.

Although a number of additional cases of tuberculosis developing within the esophagus itself have been added in succeeding years, this type of involvement is still extremely rare, and the esophagus enjoys great immunity to the direct implantation of the tubercle bacillus, particularly in the absence of any pre-existing lesion of the esophageal mucosa.

The rarity of tuberculosis of the esophagus is shown by the fact that, in 1,400 autopsies on tuberculous patients performed in the 10 years between 1932 and 1942 at Bellevue Hospital, there was only 1 case of tuberculosis of the esophagus. In addition to this isolated case, Carr and Spain¹³ described a case of a tuberculous lesion apparently engrafted upon a primary carcinoma of the esophagus.

A somewhat similar case of tuberculosis developing within a carcinoma of the esophagus is described by Dean and Gregg.¹⁴

There is no roentgen alteration in the appearance of the esophagus specifically ascribable to an intrinsic tuberculous lesion.

A well confirmed case of tuberculosis of the esophagus that had been radiographed was presented by Presser¹ before the Vienna Roentgenological Society in 1934. A roentgen study of the esophagus was made in a female, aged 63, because of dysphagia. This revealed a moderate degree of narrowing of the lumen at about the midportion with some delay in the passage of the opaque material. When the esophagus was completely filled, the contour of the narrowed area was serrated due to nichelike projections. When only partially filled, there were many small rounded intraluminal filling defects. On the basis of this evidence a diagnosis was made of an ulcerating stenosing infiltration of the middle of the esophagus. Autopsy in the Allgemeinen Krankenhaus revealed an "oesophagitis tuberculosa ulcerosa." There were many ulcers, some of them confluent. The walls of the ulcers were considerably undermined.

curring spontaneously. Stimulation of the vagus nerve however, caused further upward ascent of the stomach with an exaggeration of the hernia.

An interesting experimental observation was that reflex shortening of the esophagus occurred almost invariably from stimuli in the upper abdomen of the experimental animal.⁸ This response of the esophagus occurred after mechanical stimulation of the peritoneum by scratching with a pointed instrument. Among other causes were manipulation of the liver, traction on the gallbladder or overdistention of the viscus following the injection of normal saline solution. All of these reflexes were abolished by section of the vagus nerve or after the administration of atropine.

2 In a review of 220 cases of short esophagus with partial thoracic stomach by Olsen and Harrington⁹ only 4 per cent appeared to be of congenital origin. In most cases an actual esophagitis was present, which, as a result of cicatrization, had produced traction upon the stomach thereby drawing it up into the chest. In 36 cases there was a preceding history of excessive vomiting. Of these 19 patients had an obstructive duodenal ulcer. Allison, Johnstone and Rovee¹⁰ also emphasized the fact that in many cases ulceration and fibrosis may be the important factors in causing a shortening of the esophagus with traction upon the stomach producing a herniation through the hiatus.

Caution must be exercised in connection with the existence of islands of gastric glands at the junction of a shortened esophagus and a thoracically placed stomach. While there is considerable evidence to show that such aberrant rests may be of congenital origin, another factor may operate in the case of an already established esophagitis with ulceration. The upward traction of the scarred and contracted esophagus by pulling on the cardiac end of the stomach may cause some displacement and isolation of nests of gastric glands within the area of constriction. These may be interpreted as

having been the primary cause of the inflammatory reaction, when as a matter of fact they may actually be a secondary result of the pathologic process itself with displacement of isolated islands of gastric glands.

Perhaps the only cases of congenitally aberrant gastric glands which may be acceptable are those present in an otherwise normal esophagus or at such a distance proximal to the area of inflammatory stenosis as to preclude the possibility of their having been originally drawn up from the stomach itself.

ROENTGEN DIAGNOSIS

The roentgen diagnosis of this condition depends on the demonstration of three outstanding factors:

- 1 The appearance of the stomach in the chest cavity above the diaphragm.

- 2 The delineation of the definitely shortened esophagus joining the stomach within the thorax.

- 3 The demonstration of definite rugal folds within the "thoracic stomach."

In the differential diagnosis, the possibility of a true herniation of the stomach must be considered. The roentgen findings in gastric herniation will be discussed in greater detail later. Suffice it to say here that in this condition the esophagus retains its normal length. However, one must carefully guard against the fact that in true herniation of the stomach, the lower portion of the esophagus may be carried up with it into the chest cavity. The lower portion of the esophagus will then appear kinked on itself and somewhat tortuous. One will obtain the impression that if the hernia could be reduced, the entire esophagus would straighten out and would appear normal in length, making its exit below the diaphragm.

Clinically, such differential diagnosis is of great importance from the standpoint of possible surgical intervention. While a true

hernia of the stomach may be reducible, obviously the "short-esophagus" type of abnormality does not lend itself to such treatment

Another possibility is that of an ampullary dilatation at the cardiac end of the esophagus. This may arise as a result of temporary failure of relaxation at the cardia. Those other factors that enter into the formation of the ampulla have been described previously in Chapter 3. The features that will aid in differential diagnosis are (1) the inconstancy of this finding during the course of a single examination or on repeated examination, (2) a study of the mucosal relief will fail to show the presence of the rugal structure characteristic of the stomach.

On careful examination a diverticulum

involving the distal area of the esophagus will show the presence of a neck communicating with the esophagus above the extreme cardiac end, which will be traced entering directly into the stomach. Moreover, mucosal folds are never found within the confines of a diverticular sac.

Illustrative Cases The following cases are illustrative of "thoracic stomach" or so-called herniation of the stomach of the short-esophagus type.

E. L., female, aged 60. This patient gave a history of difficulty in swallowing, associated with substernal pain of 6 years' duration. At times, a piece of food appeared to get stuck substernally, following which she would be unable to eat for a few minutes. Occasionally, she regurgitated solid food and blood-streaked material. During these years there was no exaggeration of the symptoms.



FIG. 110 (A, Left) "Thoracic stomach" with diverticulum of the esophagus (B, Right) Same patient. Detailed appearance of the diverticulum of the short esophagus and the "thoracic stomach."

There were fluctuations in weight, so that weight lost at one time was recovered later on.

Roentgen examination (Fig 110 A and B) showed a short esophagus narrowed at its junction with a "thoracic stomach." The esophagus showed a diverticulum originating from the anterior wall of its midportion.

This association of 'thoracic stomach' and esophageal diverticulum brings up three possible explanations: (1) since the thoracic stomach may be a congenital anomaly, that the diverticulum of the esophagus is also of congenital origin in this case; (2) that the diverticulum of the esophagus is pulsion in origin, as the result of obstruction at the distal end of the esophagus over a period of many years; or (3) that the association is entirely coincidental in nature.

I am inclined to the opinion that the relationship is purely coincidental for the following reasons: (1) diverticula of the esophagus are comparatively common although "thoracic stomach" is rare; (2) the location of the diverticulum on the anterior wall near the bifurcation of the trachea is

the common site of diverticula of the traction variety; and (3) the pulsion origin of the diverticulum, as the result of obstruction at the distal end of the esophagus, is not definitely tenable, since in those cases of considerably more marked obstruction of the esophagus as in "cardiospasm," the presence of a diverticulum of the esophagus is rare.

Another example of "thoracic stomach" is as follows:

M. B., female, aged 47. The chief complaint of this patient was difficulty in swallowing for about 1 year. In addition, the patient complained of substernal pain aggravated by food. She was able to swallow liquid and soft foods easily but not bulky foods. These seemed to stick subternally.

Roentgen examination revealed a short esophagus, associated with a 'thoracic stomach' (Fig 111 A). There was narrowing of the esophagus at its junction with the stomach. Figure 111 B shows the 'thoracic stomach' with the characteristic rugal structure.

The patient's symptoms became progressively worse. Pain and burning appeared in



FIG 111 (A, Left) 'Thoracic stomach' with a short esophagus (B, Right) Same patient. Note the rugal folds within the 'thoracic stomach.'

the epigastrium promptly after the ingestion of solids or liquids. Two years after the original roentgenograms, she was referred to the hospital from the outpatient clinic. She had lost 30 pounds in the preceding year and vomited frequently. Esophagoscopy was reported as revealing an annular mass with definite ulceration at the cardiac end. While on the ward she developed a spreading infection from an acute parotitis.

At autopsy, in addition to a bilateral suppurative parotitis and coronary atherosclerosis, there was a stricture of the esophagus.

The microscopic examination revealed the following findings: "Section shows a greatly thickened wall and a poorly preserved mu-



FIG 111 C Same patient as is shown in Figure 111 A and B. Shortly before death, showing marked constriction at the junction of the esophagus and the "thoracic stomach."

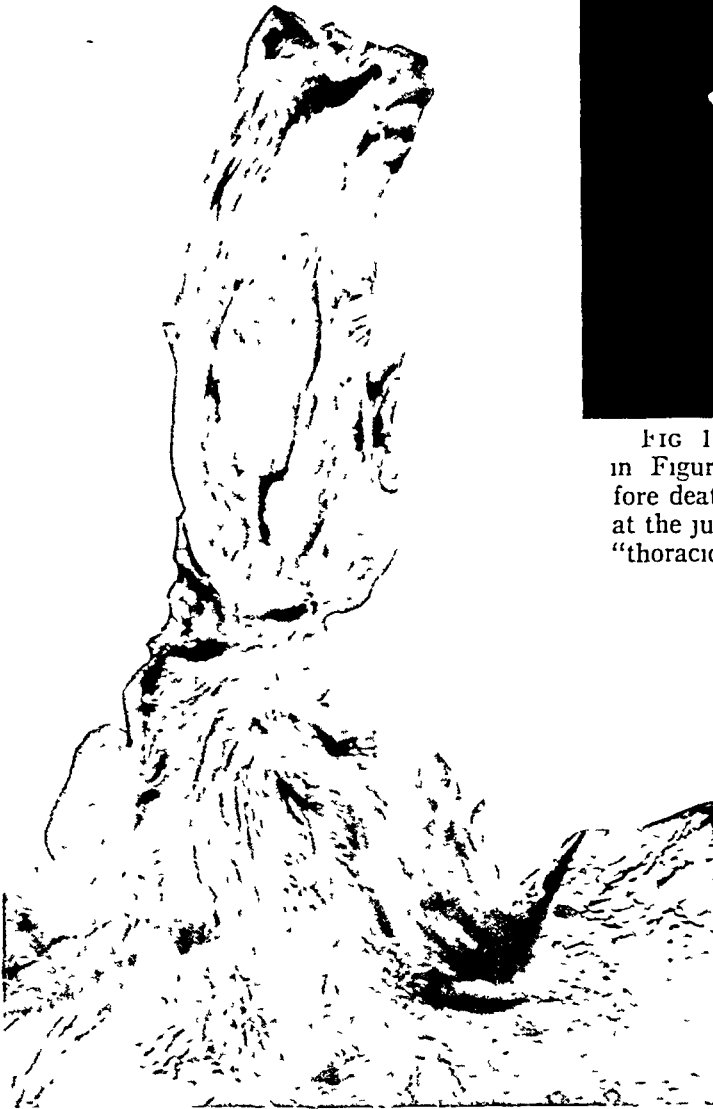


FIG 111 D Same patient as is shown in C. Appearance of the esophagus at autopsy, with an ulcer proximal to the strictured area.

cosa, which is absent completely, except for one small cluster of stratified squamous epithelial cells. The latter appears normal and is confined to the basement membrane. In the central portion of the section it is thickest and here there is almost no recognizable remaining muscle tissue. The wall is composed of fibrous tissue which, in most areas is very dense with hyalinized collagen fibers while in other areas there is a more cellular connective tissue, with fibroblasts and occasional clusters of lymphocytes and plasma cells. The blood vessels are congested in certain areas and in others are obliterated by fibrous tissue. There are no epithelial structures in the wall. In adjacent areas of the esophagus there are scattered areas of fibrosis and the muscle wall appears to be hypertrophied in one area.

The diagnosis was "Chronic inflammatory tissue with marked fibrosis."

Roentgenographic examination shortly before death (Fig. 111 C) showed a marked increase in the degree of constriction of the esophagus at its junction with the stomach. The contour, however, was quite smooth and faded gradually into the normal wall. The pre autopsy roentgen diagnosis was benign stricture which had become more accentuated since the original examination 2 years before. There was no evidence of a niche in the roentgenogram of the esophagus. In the autopsy specimen, however, there was definite evidence of an ulcer proximal to the zone of constriction. This region was apparently obscured by the barium above the constriction.

Figure 111 D shows the appearance of the esophagus as noted at autopsy with an ulcer proximal to the strictured area.

A well documented case of an ulcer of the esophagus associated with a thoracic position of the cardiac end of the stomach is the following:

F V, male, aged 54. This patient gave a 20 year history of duodenal ulcer. During the preceding 3 years he also developed dysphagia particularly for solid foods. There was no significant weight loss. Esophagoscopy revealed several areas of ulceration in the lower third of the esophagus with a definite stricture through which bougies up to No. 24 were passed. The lower end of the esophagus was fixed in the area of stricture. A diagnosis of benign stricture was made. Because of the fact that the patient also had a duodenal ulcer corroborated by radiographic examination a subtotal gastrectomy was done. In addition to the resection the stricture of the



FIG. 112 Note the strictured area of the esophagus with the anteriorly placed niche due to ulceration. Note also the thoracic position of the cardia of the stomach, apparently due to the traction exerted upon it by the shortening that resulted from the inflammatory lesion of the esophagus.

esophagus was dilated. However, improvement was only temporary. The patient underwent a second operation. A transthoracic exploration of the lower third of the esophagus was done and it was found to be surrounded by a mass of inflammatory tissue. A plastic procedure similar to that used in pyloroplasty was performed to increase the lumen. Later inquiry revealed that the patient was feeling better.

Radiographic examination of the esophagus prior to esophageal surgery (Fig. 112) shows the area of constriction at its distal end where it joins a herniated portion of the stomach. Note the projection of the anterior wall just proximal to the area of constriction, presumably representing an area of ulceration. In all probability the herniation of the stomach was a secondary manifestation due to the pull exerted upon the stomach because of the scarring and the contraction of the esophagus.

REFERENCES

- 1 Keith, A The nature of the mammalian diaphragm and pleural cavities, *J Anat & Phys* 39 243, 1905
- 2 Bright, R Account of a remarkable misplacement of the stomach, *Guy's Hosp Rep* 1 598, 1836
- 3 Bailey, P A case of thoracic stomach, *Anat Rec* 17 107, 1919
- 4 Findlay, L, and Kelly, A B Congenital shortening of the esophagus and the thoracic stomach resulting therefrom, *J Laryng & Otol* 46 797, 1931
- 5 Clerf, L H, and Manges, W F Congenital anomalies of the esophagus with reference to the congenitally short esophagus with a portion of the stomach above the diaphragm, *Ann Otol, Rhin & Laryng* 42 1058, 1933
- 6 von Bergmann, G, and Goldner, M *Functionelle Pathologie*, p 75, Berlin, Springer, 1932
- 7 Rall, J E, Gilbert, N C, and Trump, R C Effect of vagus stimulation on the longitudinal fibres of the stomach and esophagus, *Quart Bull Northwestern Univ M School* 19 194, 1945
- 8 Dey, F L, Gilbert, N C, Trump, R, and Roskelley, R C Reflex shortening of the esophagus in the experimental animal with the production of esophageal hiatus hernia, *J Lab & Clin Med* 31 499, 1946
- 9 Olsen, A M, and Harrington, S W Esophageal hiatal hernias of the short esophagus type, etiologic and therapeutic considerations, *J Thoracic Surg* 17 189, 1948
- 10 Allison, P R, Johnstone, S, and Royce, G B Short esophagus with simple peptic ulceration, *J Thoracic Surg* 12 432, 1943

THE STOMACH

REFERENCES

- 1 Keith, A The nature of the mammalian diaphragm and pleural cavities, *J Anat & Phys* 39 243, 1905
- 2 Bright, R Account of a remarkable misplacement of the stomach, *Guy's Hosp Rep* 1 598, 1836
- 3 Bailey, P A case of thoracic stomach, *Anat Rec* 17 107, 1919
- 4 Findlay, L, and Kelly, A B Congenital shortening of the esophagus and the thoracic stomach resulting therefrom, *J Laryng & Otol* 46 797, 1931
- 5 Clerf, L H, and Manges, W F Congenital anomalies of the esophagus with reference to the congenitally short esophagus with a portion of the stomach above the diaphragm, *Ann Otol, Rhin & Laryng* 42 1058, 1933
- 6 von Bergmann, G, and Goldner, M *Functionelle Pathologie*, p 75, Berlin, Springer, 1932
- 7 Rall, J E, Gilbert, N C, and Trump, R C Effect of vagus stimulation on the longitudinal fibres of the stomach and esophagus, *Quart Bull Northwestern Univ M School* 19 194, 1945
- 8 Dey, F L, Gilbert, N C, Trump, R, and Roskelley, R C Reflex shortening of the esophagus in the experimental animal with the production of esophageal hiatus hernia, *J Lab & Clin Med* 31 499, 1946
- 9 Olsen, A M, and Harrington, S W Esophageal hiatal hernias of the short esophagus type, etiologic and therapeutic considerations, *J Thoracic Surg* 17 189, 1948
- 10 Allison, P R, Johnstone, S, and Royce, G B Short esophagus with simple peptic ulceration, *J Thoracic Surg* 12 432, 1943

THE STOMACH

The Normal Stomach

TECHNIC OF ROENTGEN EXAMINATION

Before beginning fluoroscopic examination of the alimentary tract the heart and the lungs are scanned in routine fashion. Even though the primary purpose of the examination is to determine the presence of any abnormality of the alimentary tract such visualization of the chest may offer information of considerable value. In the first place lesions of the heart and the lungs of a coincidental nature may be found. Secondly, some of the pathologic findings discovered in this manner may throw considerable light upon the abdominal symptoms, for the investigation of the cause of which the examination was primarily undertaken.

Thus an unsuspected lesion of the lung of a tuberculous nature may account for the patient's dyspepsia and loss of weight. An aneurysm of the aorta or an enlarged heart may explain distress vaguely localized to the upper abdomen. Moreover, direct evidence of the presence of a viscus within the chest may also be discovered, particularly the presence of herniation.

The freedom of mobility of both diaphragms should be determined as well as their relative position. Abnormalities of diaphragmatic behavior may be discovered which may be responsible for the abdominal symptoms. Marked elevation of the diaphragm may be due to atrophy or to upward displacement.

On completing this routine inspection of the chest, the abdomen itself should also be studied with considerable care. Opaque

shadows due to urinary calculi may be noted and occasionally even calcified processes in the gallbladder, the liver or the spleen may be observed. In addition, various other evidences of calcification may be present, due to lymph nodes, phleboliths and cysts. The amount of gas throughout the alimentary tract can be observed, and in exceptional cases the diagnosis of intestinal obstruction may be made thereby in an individual in whom this condition had not been suspected clinically.

Direct visualization of the air bubble of the stomach may yield important findings. Instead of the normal contour, it may be deformed by encroaching shadows due to new growths.

After this superficial investigation as just outlined, one is prepared to continue with the more detailed investigation of the stomach itself.

Under ordinary circumstances barium sulfate is best administered suspended in water. The patient is given a glassful of equal parts of barium and water thoroughly stirred just before ingestion. A suspension of this kind will enter small areas much more readily than if the barium were mixed with food or made too thick in any other way. Moreover this thin fluid medium leaves the stomach more readily by way of the pylorus and the prompt visualization of the duodenum and the small intestine is aided thereby. For special studies there may be modifications in the preparation of the barium suspension.

The examination is best conducted in the morning, the patient having been instructed to abstain from food and drink since the

pyloric antrum was a definite structure anatomically preformed and possessing a sphincter antri Kastle, Rieder and Rosenthal⁶ devised a biroentgenograph for making a large number of exposures during a single respiratory pause, each exposure being reduced to a small fraction of a second This enabled them to study the course of peristaltic activity without the aid of fluoroscopic observation Through the application of roentgen cinematography, they showed that the antrum of the stomach varied from moment to moment This constant alteration of contour proved that the antrum was the result of gastric function and not of rigid anatomic formation These peristaltic waves were found by Kaufmann and Kienbock⁷ to have an average duration of about 21 seconds There was no evidence of any relationship of peristalsis to either gastric acidity or cardiac activity

THE STOMACH IN INFANCY

Lewis⁸ examined five stomachs from embryos ranging between 10 mm and 45 mm in length In the youngest embryo (10 mm) the stomach is already divided into an expanded pars cardiaca and a tubular pars pylorica Between these two areas is the incisura angularis In the 16-mm embryo the body of the stomach may be recognized along the lesser curvature, separating the esophageal cone from the incisura angularis By the second month of fetal life the fundus may be well developed

There is no definite normal type of stomach in the newborn, since this organ may show considerable variation in contour in different infants and in the same infant at different times⁹

These findings were corroborated by Bouslog and his associates¹⁰ in the roentgenographic examination of 133 infants varying in age from 1 week to 6 months There was considerable variation in both the size and the shape of the stomach within normal limits Moreover, barium reten-

tion in the stomach after 8 hours was noted occasionally in the apparently normal infant's stomach

THE TONUS OF THE STOMACH

Tone is that power of the stomach which enables its wall to enfold the gastric contents and maintain it in a more or less uniform column Tone, therefore, has a definite influence upon the form of the stomach In fact, Schlesinger¹¹ originally believed that differences in form depended entirely upon concomitant variations in tone He distinguished four types of stomach (1) hypertonic, (2) orthotonic, (3) hypotonic and (4) atonic

That tone plays an important role in affecting gastric form was demonstrated experimentally by the work of Klee¹² with decerebrated cats Under these conditions the stimulating effect of the vagus and the sympathetic nerves is greatly enhanced Increased stimulation of the vagus nerve caused contraction of the stomach and active peristalsis, with increased emptying of the stomach and concomitant variations in shape

Gastric tone is therefore dependent upon two factors: (1) the peculiar structural anatomy of the stomach, and (2) the state of vagus and sympathetic activity

Hypertonicity Hypertonicity has a definite effect in altering the shape of the stomach Increase of tone may be determined by judging the distance between the lesser and the greater curvatures at the cardiac end and by comparing it with the distance between two similar points in the pyloric region In the case of normal tonicity, the distance measured at the cardia and at the pylorus is essentially the same In hypertonicity the distance between the two curvatures gradually diminishes from the cardia to the pylorus

Hypertonicity may not only manifest itself by a change in the shape of the stomach but may also be demonstrable through alteration in the roentgen appearance of

the gastric mucosa. Through the activity of the muscularis mucosae there may be an increased prominence of the rugal folds. This effect upon the 'relief' picture may show itself in two ways (1) by an increased toothlike irregularity of the greater curvature, and (2) by an exaggeration of the folds throughout the rest of the stomach. This appearance may closely simulate that seen in the presence of organic disease.

Atony. In gastric atony, if the distance between the lesser and the greater curvatures beginning at the cardia is traced distally, it is found to increase so that by the time the pylorus is reached this distance between the curvatures shows a considerable exaggeration. As barium enters an atonic stomach, it sinks to the lower pole, where it spreads out along the greater curvature without being maintained in a vertical column reaching to the cardia, as is noted in the case of normal or increased gastric tone. With diminished tone the lower pole of the stomach sags in a baglike manner. Owing to the poor tone, the pyloric portion of the stomach is visualized with considerable difficulty. Either the ingestion

of a considerable amount of barium may be essential, or upward displacement of its contents by manual palpation may be required. Compression of the barium toward the pyloric ring may also be necessary in order to force it into the duodenum so that that region may be visualized. The emptying time of the stomach may be prolonged not infrequently beyond the 6 hour limit for normal function.

However, the elongated, baglike appearance of the stomach is differentiated readily from gastric enlargement due to pyloric stenosis by the following characteristics:

1 The gastric enlargement is downward toward the pelvis in atony, and practically never does it extend considerably to the right of the midline as in organic pyloric stenosis.

2 The contour of the pyloroduodenal region may be shown to be perfectly normal in the case of functional atony, whereas in pyloric stenosis irregularity or abnormality of contour in this region may be demonstrable.

3 While there is a delay in the emptying of the atonic stomach, it never reaches the



FIG. 113 Transverse hypertonic type of stomach in a hypersthenic individual



FIG 116 (*Left*) Atonic stomach in an asthenic individual



FIG 117 (*Right*) Hypermobility of the stomach, with upward displacement in the prone position



FIG. 118 Volvulus of the stomach with permanent displacement to the right

Stokes¹⁶ at operation found the stomach and the transverse colon in a right inguinal hernia. There had been no previous clinical or radiographic indication of this unusual condition.

The stomach occupying a large scrotal hernia was noted at autopsy by Siegmund¹⁷. In none of the cases described so far had there been any radiographic studies. Lust¹⁸ showed an excellent illustration of the barium-filled stomach within a left scrotal hernia. The lower pole of the stomach was at the level of the middle-portion of the femur but was readily reducible into the abdominal cavity as determined roentgenologically. In Hartley's case¹⁹ a diverticular-like prolongation of the stomach was present in a left inguinal hernia as demonstrated roentgenologically. Cave's patient²⁰ was also radiographed. The stomach was present in a left femoral hernia.

Figure 118 illustrates volvulus of the stomach with rotation to the right. This appearance was persistently present throughout prolonged fluoroscopic examination as



FIG. 119 Cascade stomach (A *Left*) Appearance in the erect postero anterior position at 90° (B *Right*) Appearance in the erect right oblique position clearly showing the overhanging cardiac locule

well as at repeated observations at later periods

An important alteration in the configuration of the stomach is that known as the cascade stomach, in many cases this is apparently associated with the pressure of a gas distended colon. The mechanism involved is discussed under functional disorders of the colon. An example of a cascade stomach produced by an overhanging cardiac locule is shown in Figure 119 A and B. Figure 119 A is the appearance in the erect postero anterior position at 90° . The overhanging character of the cardiac locule is shown more clearly in the erect right oblique position (Fig. 119 B). A marked example of a typical cascade stomach is shown in Figure 120. Note the cup and spill type of stomach with the pyloric arm and the duodenal bulb directed downward and to the right. Note also the enormous gaseous distention of the colon in direct proximity to the stomach. The pa-

tient, J. A., aged 46, gave a history of marked constipation for 2 years and occasional pain after eating. After taking a laxative he obtained relief.

That the typical cascade stomach is the result of pressure of a distended colon is shown in a number of ways

- 1 By visual demonstration of the association of the two conditions
- 2 By the disappearance of the cascade deformity if the stomach is overdistended with barium, so as to overcome the pressure of the distended colon
- 3 By the disappearance of the cascade deformity if the colon is emptied of its gas after the administration of a cathartic or after an enema

4 By the fact that, by artificially distending the colon with air, a cascade deformity of the stomach can be produced

A bizarre type of "cascade" stomach is shown in Figure 121 A. The pars cardica is actually at a lower level than the pyloro-

duodenal region There was considerable gaseous distention of the splenic flexure That this appearance was of a transient nature and not due to any intrinsic organic disease is shown by the fact that on re-examination 1 week later, the anatomic position of the stomach is essentially normal (Fig 121 B) When it is suspected that an abnormal position of the stomach is due to "cascading" and is associated with gaseous distention of the colon, one may prove this point in two ways first, by administering additional quantities of barium to overcome the pressure exerted by the colon and noting the fact that the stomach assumes a normal position, secondly, by the preliminary administration of castor oil the night before examination to diminish the amount of gas present in the colon Or we may combine both these procedures in a restudy Not uncommonly I have seen strange abnormalities in the position of the stomach yield to the application of these procedures, with a return to the normal, thereby disclosing the nature of the mechanism involved

DUPLICATION OF THE STOMACH

Blásius in 1677 (*ibid*) described a case of a "double stomach"

In a dissected cadaver of an individual 35 years of age who during life had been troubled with continuous vomiting for a long time we found a double stomach The first part which continues from the esophagus is distinguished from the other which communicates with the intestine At the place where the two cavities were separated the lumen was very narrow so that one could barely introduce the little fingers through it This narrowed zone I conjecture was the cause of the frequent vomiting of the patient

The findings are illustrated by a drawing (Fig V, Plate VI, p 113, Blásius, G *Observationes Medicae Rariores*, 1677)

A case of duplication of the stomach was described by Ladd and Gross²¹ Radiographic examination showed a smooth, concave indentation of the greater curvature produced by the pressure of the duplication In some cases the only roentgen evidence of reduplication of the stomach may be an indeterminate type of pyloric deformity with partial obstruction^{22 23}

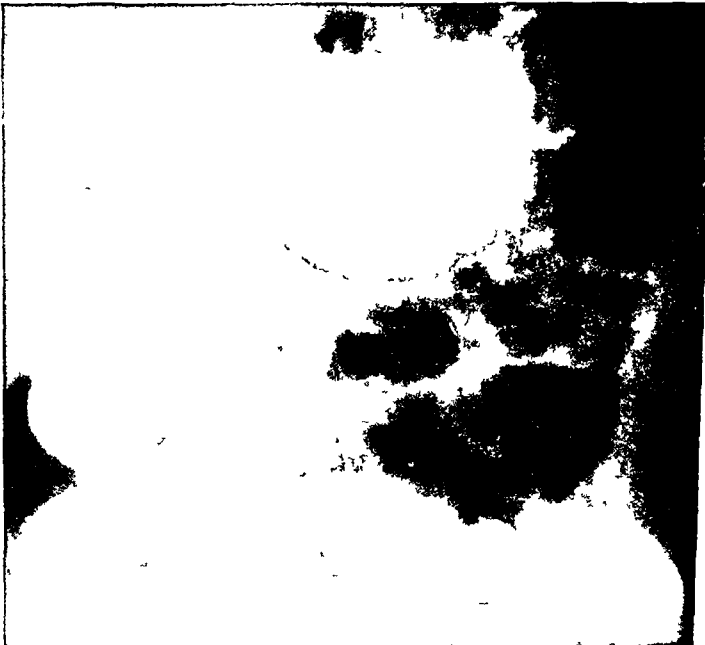


FIG 120 Cascade stomach Note the marked gaseous distention of the splenic flexure

DISPLACEMENT OF THE NORMAL STOMACH BY EXTRAGASTRIC TUMORS

Extragastric tumors may displace the stomach in almost any direction. The rela-

tion of tumors of pancreatic and of splenic origin to the position of the stomach will be discussed in later chapters dealing with lesions of the pancreas and the spleen.

Illustrative Cases A retroperitoneal



FIG. 121 (A *Top*) Note this bizarre type of cascade stomach (B *Bottom*) Examination 1 week later, showing an essentially normal appearing stomach



tumor may displace the stomach to the right

M S, female, aged 9 The child gave a 3-year history of recurring attacks of epigastric pain, which was continuous for 24 hours There was occasional vomiting with the attacks She weighed 50 pounds Physical examination revealed a large mass in the left upper quadrant Operation disclosed a tumor apparently originating from the left adrenal gland Complete removal could not be accomplished

Pathologic examination of biopsy medullary carcinoma of the adrenal gland

Roentgen examination (Fig 122) showed a large mass displacing the stomach to the right and the loops of small intestine in a downward direction There was no evidence of any intrinsic involvement of the stomach The mass was obviously of extragastric origin As noted above, the tumor was a malignant lesion of the adrenal gland

An unusual cause of upward displacement of the stomach is a cyst of the omentum

Gairdner²⁴ in 1851 gave the first description of a cyst of the omentum The cyst was found in a woman at autopsy, beneath the anterior layer of the greater omentum, and consisted of a highly transparent, closed sac between 3 and 4 inches in length and from $\frac{1}{2}$ to $1\frac{1}{2}$ inches in breadth It was lobulated in appearance The fluid in the sac was a transparent, colorless serum No ova or parasites were present He considered it as very probably a variety of simple serous cyst

The next recorded case is by Bantock²⁵ in 1881 This was a large thick-walled cyst which was present in the great omentum of a woman aged 58 A drawing accompanies the presentation, showing the cyst enclosed by the layers of the omentum



FIG 122 Displacement of the stomach by a carcinoma of the adrenal gland

The case was presented by his associate, Dr Doran, who had assisted at the removal of the cyst. In addition, he illustrated this condition by exhibiting a Hunterian specimen of a small cyst entirely within the folds of the great omentum.

From then on there were reports of isolated cases so that Hasbrouck⁶ in 1908 was able to review 54 cases, of which he considered only 24 as acceptable. Twelve of these originated from the omentum by a pedicle and were not true cysts within the layers of the omentum. He added a case of his own, a cyst originating from an endometrioma of the omentum.

Additional cases were reported by Dowd⁷ in 1911 and Pybus⁸ in 1917. Pybus' case was in a child of 4. The photograph of the patient shows a hugely distended abdomen. At operation a thin-walled cyst was

situated in the great omentum. At its upper pole it had to be peeled from the greater curvature of the stomach in order to be freed.

By 1935 Horgan⁹ had found 97 acceptable cases in the literature. The condition is apparently more common in children, 35 per cent having occurred in the first decade and 68 per cent during the first 30 years. Horgan's case of omental cyst occurred in a child 12 years old.

There is a paucity of descriptions of the radiologic findings in this condition. In Birnkrant's case radiographic examination showed marked downward displacement of the transverse colon.¹⁰

In the following case, the stomach was displaced upward by an omental cyst.

M. S., female, aged 2½ years. During the preceding 3 months, the child had passed

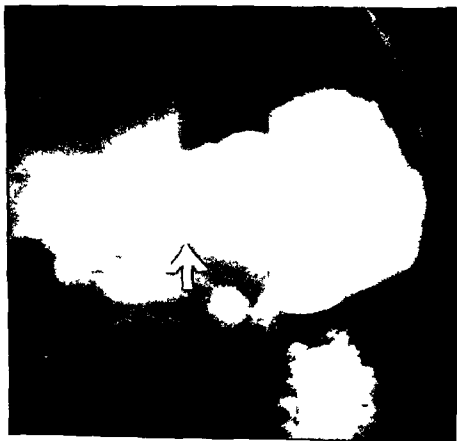


FIG. 123 A Upward displacement of the stomach by a cyst of the omentum.

bright-red blood by rectum on five or six occasions, this was accompanied by diarrhea. One month later she complained of pain in the back, which gradually became worse. Two

days before admission to the hospital the pain in the child's back was accompanied by persistent vomiting, fecal in odor. The next day the pain shifted to the abdomen.



FIG 123 B Same patient as is shown in A. Appearance of the resected omental cyst.

Physical examination revealed a mass which felt cystic just to the left of the mid line of the abdomen

At operation there was a large cyst in the greater omentum, the size of a grapefruit. There was an extension of the mass which contained numerous small grapelike cysts. The omental cyst created a pressure defect on the inferior border of the stomach and was situated near the transverse colon. The cyst was excised.

The pathologic diagnosis was "Lymphoma with cyst formation."

Roentgen examination (Fig 123 A) showed upward displacement of the stomach with a smooth pressure defect, indicating the presence of an extragastric mass as the cause. The tumor was believed to be a cyst either of mesenteric or of omental origin. The appearance of the cyst is noted in Figure 123 B.

Another clinically important cause of displacement of the stomach is aneurysm of the abdominal aorta. Aid in the roentgen recognition of this condition may be obtained from (1) evidence of partial calcification in the wall of the mass, which as a rule is globular in appearance, (2) the close anatomic relationship of this mass to the position of the abdominal aorta, and (3) evidence of erosion of the vertebrae due to pressure of the aneurysm. Because of the fibrocartilaginous nature of the intervertebral disks, they resist destruction, and the pressure of the aneurysm is exerted primarily on the anterior portion of the bodies of the vertebrae, producing irregularly outlined, concave depressions. The eleventh and the twelfth dorsal and the first and the second lumbar vertebrae are most commonly involved in the erosive process, (4) when a globular mass in anatomic relationship to the abdominal aorta shows a combination of calcification of the rim and vertebral erosion, the diagnosis of aortic aneurysm may be made with assurance.

Kampmeier³¹ noted that evidence of pressure erosion of the vertebrae was the most important finding in aneurysm of the abdominal aorta. Of 32 cases radiographed, a roentgen study of the spine was made in 24, and in 18 of these (75 per cent) erosion of the vertebrae was present. Four of this

group showed gastric displacement by the aneurysm as well as erosion, and in 1 of these 4 cases a gastric deformity was produced by the pulsating mass. The most commonly eroded vertebrae were the twelfth dorsal and the first lumbar.

The frequency of erosion of the vertebrae in abdominal aneurysm was also shown by Hubeny and Pollack,³² Elison and McNamee,³³ Scott³⁴ and Pratt Thomas.³

Displacement of the stomach by an abdominal aneurysm may be noted in the following examples.

An aneurysm of the abdominal aorta may displace the stomach to the left. The patient, J. N., aged 44, was hospitalized because of intermittent upper abdominal pain of brief duration and a small pulsating abdominal mass, which he had noted for several years. Physical examination revealed a firm, pulsating mass with thrill and bruit, present in the epigastrium and measuring about 8 x 10 cm. The Wasserman test was negative. An operation was done with wiring and electrothermic coagulation of an abdominal aortic aneurysm. Approximately 100 feet of wire was introduced into the aneurysm.

Roentgen examination (Fig 124) showed displacement of the stomach to the left by a circular mass with a calcified rim. A diagnosis of aneurysm of the abdominal aorta was made based on the following features: (1) the rounded well circumscribed character of the mass, (2) its location in relation to the anatomic position of the aorta, and (3) particularly, the calcification of the wall.

Marked displacement of the stomach to the left by an abdominal aneurysm as well as other classical features of its presence is illustrated by this fully controlled case.

J. W., male, aged 66. During the preceding 2 years the patient had complained of dyspnea. Three weeks before admission to the hospital he had had a sudden onset of dull pain in the right lower chest and in the right upper quadrant transmitted to the back. He had lost weight but did not know how much

Examination of the abdomen revealed a pulsating mass with systolic thrill and murmur in the epigastrium

The autopsy report was as follows "In the descending portion of the aorta at the level of the attachment to the diaphragm there is a sacular swelling approximately 10 cm in diameter The aorta passes anteriorly to this and at the level of the first lumbar vertebra, there is a 5-cm longitudinal defect in its posterior wall, from which the sac originates Posteriorly, the sac is intimately connected to the eleventh and twelfth thoracic and the first and second lumbar vertebrae, which have been irregularly eroded to a maximum of 2 cm The sac is filled with clotted blood The surface is flexible but here and there contains calcified atheromatous plaques On the right anterior surface, there

is a defect leading into the right pleural cavity, which is filled with clotted blood The esophagus and stomach are normal "

The final diagnosis was syphilitic aneurysm of the aorta with rupture of the aneurysm and hemorrhage into the right pleural space

Roentgen examination of the stomach (Fig 125 A) revealed evidence of considerable pressure upon the lesser curvature of the proximal portion of the stomach with considerable displacement to the left The stomach itself showed no evidence of any intrinsic organic disease The cause of the displacement was best seen in the lateral film (Fig 125 B), which showed a fairly well-rounded, sacular area There was an

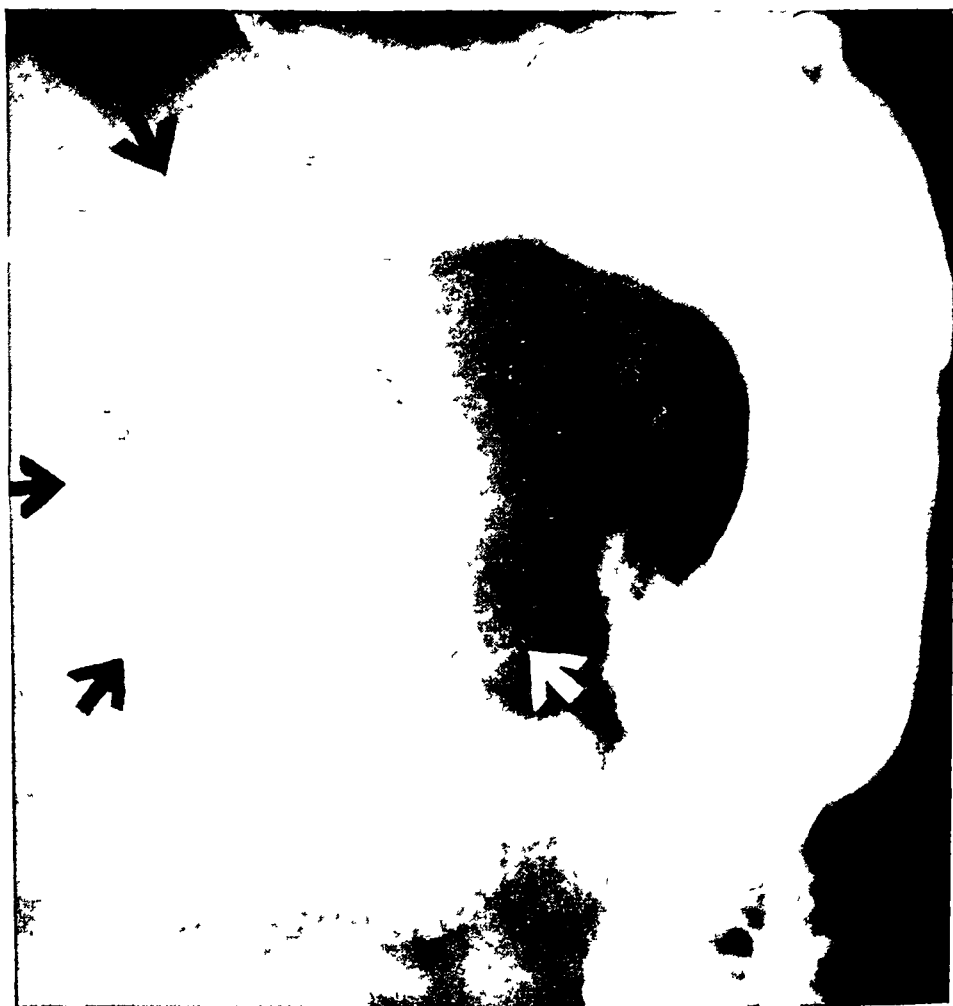


FIG 124 Displacement of the stomach by an aneurysm of the abdominal aorta
Note calcification in the rim of the aneurysm



FIG 125 (A *Left*) Displacement of the stomach by an aneurysm of the abdominal aorta (B, *Right*) Same patient. Note the erosion of the vertebrae, produced by the aneurysm, as well as the calcification of the wall

irregular distribution of calcification in the rim of this mass. Note also the erosions of the vertebrae produced by the pressure of the aneurysm.

The diagnosis of an abdominal aneurysm as the cause of the displacement of the stomach not only depended on its location and its rounded contour and calcified rim but received further support from the fact that the lower dorsal and upper lumbar vertebrae were eroded.

The retrogastric pressure of an abdominal aneurysm may distort the appearance of the stomach in such a way as to simulate the deformity of an intrinsic malignant lesion.

T. M., male, aged 63. The patient complained of substernal pain and, at the time of admission to the hospital, was suffering from

a severe psychosis with hallucinations and disorientation. At another institution it had been found that he was a luetic, with apparently luetic heart disease and thoracic aneurysm. The clinical diagnosis was syphilitic aortitis with thoracic aneurysm.

On abdominal examination, while no masses were palpable, there was a marked pulsation in the epigastric region synchronous with the heartbeat. At autopsy, in addition to atheromatous changes, there was a huge aneurysm where the thoracic aorta pierced the diaphragm. This aneurysm involved 18 cm of the aorta and was 15 cm in width. It filled most of the pleural sac. In the proximal portion of the abdominal aorta, there was another aneurysm 5 cm in width and pouching out for a distance of 4 cm. In the distal portion of the abdominal aorta there was another aneurysm, 7 cm in length and 5 cm in its widest portion.

The report of the examination of the stomach was as follows: "The stomach is



FIG 126 Deformity of the stomach by pressure from an aneurysm of the aorta

small and its rugae are prominent. The mucosa is completely smooth and there are no intrinsic lesions."

Roentgen examination of the stomach (Fig 126) showed an irregularly outlined, translucent area occupying the pars media. Below this was a wide, comparatively smooth defect of the greater curvature. (Careful fluoroscopy was not possible because of the patient's serious condition.) Based on the roentgen evidence, a diagnosis of malignancy of the stomach was made. As noted at autopsy, the deformity was due to the localized area of pressure of the abdominal aneurysm upon the posterior wall of the stomach.

When anterior displacement of the stomach by a retrogastric mass is suspected, the roentgen examination is best conducted with the patient in the left lateral position, supine or erect, without and with the administration of barium by mouth. With the patient on his back and a 14 x 17 cassette tangential to his left side, the ray is directed from right to left. An essentially similar procedure is employed when the patient is erect. At times in the film taken without administration of barium, a soft tissue mass may be noted close to the spine. However, such observations always should be supplemented by examination in the same position after the stomach has been filled with barium.

Ingel and Lysholm¹¹ in their study after the administration of an effervescent mixture believed that any increase in the distance between the posterior wall of the stomach and the spine of more than the width of a vertebra was suggestive of an intervening retrogastric mass. It is important to realize, however, that anatomically the stomach is directed obliquely from the posteriorly placed cardia to the more anteriorly placed pylorus. There is, thus, a gradual increase in the distance between the spine and the stomach from the cardia to the pylorus. This increase in the distance may be further enhanced by the habitus of the patient and may be much more marked in the hypersthenic and the sthenic types of individual.

Therefore all these factors must be kept

in mind in determining whether there is any actual forward dislocation of the stomach by a retroperitoneal mass.

Of greater significance is the evidence of an actual pressure defect on the posterior wall. The diagnosis of a retrogastric tumor will then depend (1) on the actual demonstration of a soft tissue mass in the left lateral films of the abdominal cavity in the region of the spine, (2) displacement of the barium or gas filled stomach, particularly in association with (3) evidence of an actual pressure deformity on the posterior wall.

The exact nature of the retrogastric tumor cannot be determined by this procedure. While it may be due to a tumor of the body of the pancreas, similar findings may be obtained when the lesion is a retroperitoneal sarcoma or a tumor of renal or adrenal origin.

The role of the pancreas in causing displacement of the stomach will be discussed in a later chapter.

The pressure of a retrogastric mass on the stomach is illustrated by the following case.

J. W. male aged 65. During the preceding 5 weeks the patient had complained of dyspnea on exertion, weakness, a hacking cough and anorexia. The patient coughed up $\frac{1}{2}$ a cupful of red blood per day during the 10 days before admission to the hospital. He had lost 35 pounds.

He was a cachectic white male acutely and chronically ill, markedly dyspneic and convulsed with paroxysms of coughing. Examination revealed a mass occupying the upper abdomen. This mass was outlined by percussion only because marked voluntary rigidity prevented palpation. There was no tenderness.

The autopsy report was "Kidneys: the right kidney weighs 130 Gm. There are a few small cysts bulging on its external surface. These contain clear fluid. The surface is smooth and no unusual changes are noted. The left kidney weighs 500 Gm. The surface is roughly nodular and the nodules vary in size and shape but are of one general pattern—broad base with conical sometimes umbilicated surface. The color of these nodules varies from whitish gray to bright yellow brown. The intervening surface is gray brown.

in color and smooth. The capsule strips with difficulty and is adherent over some of the nodules. On section of the kidney, the normal architecture and markings are obliterated. The parenchyma has been generally replaced by a mottled grayish-yellow tissue found in nodules of varying sizes and also diffusely throughout the kidney from pelvis to capsule. Some of these areas are soft, red-brown and hemorrhagic. The calyces are dilated and the mucosa is thick, congested and bright yellow-green in color. The calyces and pelvis contain soft, yellow, cheesy material. The pelvico-ureteral junction appears to be blocked by a

gray-white fungating mass extending into the pelvis from the kidney parenchyma. There is a large mass of nodes at the hilus containing material similar to that found in the kidney, and there are similar smaller masses of nodes extending upward along the aorta. The mass at the hilus and that at the kidney are seen to push the stomach anteriorly. There is a solid fused mass of nodes at the hilus which completely surrounds and partially constricts the inferior vena cava. There is a small number of similar glands in the peripancreatic tissue.

"Gastro-intestinal tract. The esophagus,



FIG 127 (A, *Left*) Adenocarcinoma of the left kidney. No displacement of the stomach in the postero-anterior position. (B, *Right*) Same patient. Erect left-lateral position, showing forward displacement of the proximal portion of the stomach and a pressure defect on the posterior wall. (C, *Below*) Same patient. Examination in the left-lateral supine position, showing anterior displacement of the proximal portion of the stomach and the pressure defect of the posterior wall produced by the tumor mass.



FIG 128 (A, *Left*) Tumor of the left adrenal gland, showing displacement of the stomach to the left (B *Right*) Same patient Erect left lateral position, showing forward displacement of the stomach by the retrogastric tumor

stomach, small and large intestine show no intrinsic changes except for mild congestion of the stomach

"Pathologic diagnosis Adenocarcinoma of the left kidney with metastases to regional and distant lymph nodes, left adrenal and both lungs"

Roentgenographic examination (Fig 127 A) of the stomach in the usual postero anterior position showed no evidence of any abnormality. Examination in the erect left lateral position (Fig 127 B) showed forward displacement of the cardiac portion of the stomach, with evidence of a pressure defect. Note that the distance between the anterior wall of the vertebral column and the posterior wall of the stomach is considerably greater than the distance occupied by the body of the vertebra. It was not only the increased distance between the vertebral column and the posterior wall of the stomach but also the evidence of a pressure defect that justified the diagnosis of a retrogastric mass. This forward displacement of the stomach as well as the pressure defect were also shown in Figure 127 C with the exposure made with the patient in the supine position the cassette being placed against the left side of the abdomen and the ray directed horizontally through the abdominal wall from right to left.

This case illustrates the anterior displacement of the stomach produced by a retrogastric mass and the technical procedure employed in the demonstration of such a lesion. In this particular case the retrogastric tumor was a carcinoma of the left kidney.

Forward displacement by a tumor of adrenal origin is illustrated by the next case.

J. R. male, aged 37. The patient gave a 3 week history of pain in the left lower chest posteriorly as well as in the left upper quadrant of the abdomen. He had fever (101° F to 102° F) marked leukocytosis and a weight loss of 30 pounds.

Roentgen examination of the lungs revealed tuberculosis of the right apex. Examination of the sputum was negative for tubercle bacilli.

Physical examination of the abdomen was essentially negative except for tenderness in the left upper quadrant.

The operative findings were as follows. On opening the peritoneum there was a moderate amount of clear fluid. The liver and spleen were normal. In the left upper quadrant retro

contains a small amount of fibrous stroma and capillaries. The cells have invaded the walls of the blood vessels, but no actual penetration into the lumen is seen. In the area of tumor adjacent to the kidney there is a thickened renal capsule between the tumor and the kidney itself. There is a marked round-cell infiltration of this capsule, but no tumor cells are in it. The kidney is compressed with hyalinization of the glomeruli and marked fibrosis replacing tubules and glomeruli.

Fat stain of the tumor reveals a moderate number of fat droplets within the tumor cells, but the larger part of the cytoplasm is clear. No tissue resembling adrenal gland is seen. There are numerous areas of infiltration by hemosiderin-bearing phagocytes.

Diagnosis Carcinoma of the kidney (clear-cell type)

The rounded area in Figure 130 A was therefore the result of compression of the posterior wall of the stomach by the retrogastric carcinoma of the left kidney. The intact mucosal pattern noted in Figure 130 B aided materially in eliminating an intraluminal lesion as the cause.

THE GASTRIC AIR BUBBLE

Of considerable importance is the gastric air bubble. A moderate amount of air is normally present in the stomach, and rising to the cardia in the erect position produces the characteristic gastric air bubble. Its size and shape may show a great deal of variation because of differences in body habitus. Secondly, the amount of air at any given time will produce considerable change. This is particularly true when air is swallowed to a pathologic degree. On observing the barium being swallowed and its passage through the esophagus, small amounts of air may be noted incorporated within the barium mass. As the swallowing act continues, the amount of air reaching the stomach may distend it to a considerable degree. Rising to the uppermost portion of the stomach, the gastric air bubble becomes abnormally prominent. With the act of belching and the sudden escape of air, momentarily the air bubble may disappear completely or may show considerable diminution in size. Soon, with each successive

swallow, there is a reaccumulation of air in the stomach, with a return to the original size of the air bubble. That the belching of gas depends entirely on the eructation of air previously swallowed was shown by Leven,¹⁷ who analyzed the eructated gas and showed that it consisted entirely of air and that gastric fermentation played no role in the process.

The gastric air bubble under certain conditions may become chronically enlarged. The left diaphragm may then be displaced upward, with encroachment upon the heart and the lungs. Moreover, such large amounts of air in the stomach may be a contributing factor in the production of gaseous distention of the intestine, since gastric peristaltic activity may force some of the air onward through the duodenum. Occasionally, the normal duodenal bulb will show in the roentgenogram an air cap surmounting it, presumably derived from the air in the stomach on its way into the intestine.

Although the air bubble shows considerable variation in size and shape, it is smooth and rounded in contour. Its shape may be altered by the presence of considerable fluid in the stomach, either as a result of hypersecretion or of the ingestion of food or of the opaque suspension used in examination. In this manner, a horizontal line will mark the lower limit of the air bubble, the rest of which, however, remains dome-shaped. Near its upper region one may occasionally note a portion of the cardiac shadow.

The air bubble may be displaced by extragastric masses or by an overdistended colon. It may be overlapped by the splenic flexure. A more serious abnormality of the air bubble, with irregularity of contour, results from the intrusion of a new growth, arising either within the stomach itself or, rarely, extending into it by perforation of an extragastric mass.

An example of a huge air bubble of the stomach associated with considerable gaseous distention of the colon is illustrated by Figure 131.

GASTRIC MOTOR FUNCTION

The first attempts to determine the normal emptying time of the stomach by roentgen observation were those of Jollasse³⁸ in 1907, and particularly by Haudek³⁹ in 1912. Although there is considerable variation in the rate of emptying of the normal stomach the 6 hour period as established by Haudek, is clinically satisfactory.

Ordinarily the normal stomach will have emptied completely during that time. However the retention of small amounts of barium in the stomach beyond this time does not necessarily mean that an organic obstructive lesion is present preventing the exit of the gastric contents. Even a residue of one fourth the barium suspension may result from factors of a purely functional nature. When one half or more of the barium is retained, the probability is very strong that an obstruction of the pylorus is present, either as a result of organic stenosis or of spasm in the presence of an ulcer. Twenty-four hour gastric retention may be considered as definite indication of the presence of an organic lesion. Normal emptying of the stomach within 6 hours may occur as an expression of all of the factors involved in the determination of gastric motility. It does not preclude the possibility, however of an abnormality of one of the factors, which may be balanced by a hyperactivity of another. Thus spasm of the pylorus may be compensated for by gastric hyperperistalsis.

Abnormally rapid emptying of the stomach may occur as a result of exaggerated peristaltic activity or in the presence of a gaping pylorus, particularly due to rigid infiltration.

Of importance roentgenologically is the effect of the emotions upon the emptying of the stomach. It is not at all uncommon to examine a patient for the first time and find apparent evidence of functional derangement of tone, peristalsis and motor function. The anxiety of the patient and the strangeness of his surroundings may con-



FIG. 131. Huge air bubble of the stomach. Note also the marked gaseous distention of the colon.

tribute to this change. Re-examination at a later time when the patient's fears have been calmed and he is less apprehensive may show the motor manifestations to be more nearly normal.

GASTROSPASM

One of the earliest studies of gastropspasm was that of Holzkecht and Luger.⁴⁰ Gastropspasm may be associated with intrinsic organic disease, or it may be a secondary manifestation from an extragastric cause, such as cholelithiasis or duodenal ulcer. Autonomic imbalance may be an important factor.

Gastropspasm may be of four types: (1) Spasm may be limited to the pyloric ring. (2) A highly circumscribed type of spasm is the incisura of the greater curvature. Though occasionally of functional origin, it may be seen opposite an ulcer of the lesser curvature and also in association with malignant ulceration. In the latter condition the incisura is apt to be broader than

in benign lesions and may be due in part to actual organic involvement in the malignant process (3) Large areas of spastic involvement of the stomach may be noted, particularly in the pars pylorica This region may show marked constriction under fluoroscopic observation with, at times, complete obliteration of the entire region When filled with barium this region is smooth in contour without evidence of destruction of the normal mucosal markings This helps in differentiating the condition from malignant infiltration Moreover, the contour may show variation of outline at different times The administration of atropine to physiologic effect may possibly aid in differential diagnosis by diminishing the spastic manifestations Marked pylorospasm may not necessarily be altogether functional and in some cases it may be associated with the presence of inflammatory disease of the pylorus or may be due to the actual presence of an ulcer (4) In total gastropspasm, the entire stomach partakes in the process, and the appearance

may simulate that of a scirrhus carcinoma of the stomach The differential diagnosis lies primarily in the inconstant manifestations in spasm as opposed to the permanent character of the deformity in malignancy, as well as in the demonstration of the intact mucosal relief when the abnormality is spastic in nature Cases of total gastropspasm have been described as a result of lead colic, nicotine poisoning and arteriosclerosis of the abdominal vessels

Pylorospasm resulting in marked delay in gastric emptying may be an allergic response to food sensitivity ¹¹

Illustrative Case The alterations in the contour of the stomach produced by functional gastropspasm are demonstrated by the following case

M L, male, aged 38. This patient complained of vague gastric discomfort after meals He vomited occasionally He said that he had lost 40 pounds in the last 5 years He had undergone appendectomy 27 years previously Physical examination of the abdomen was essentially negative

Exploratory laparotomy revealed no evi-

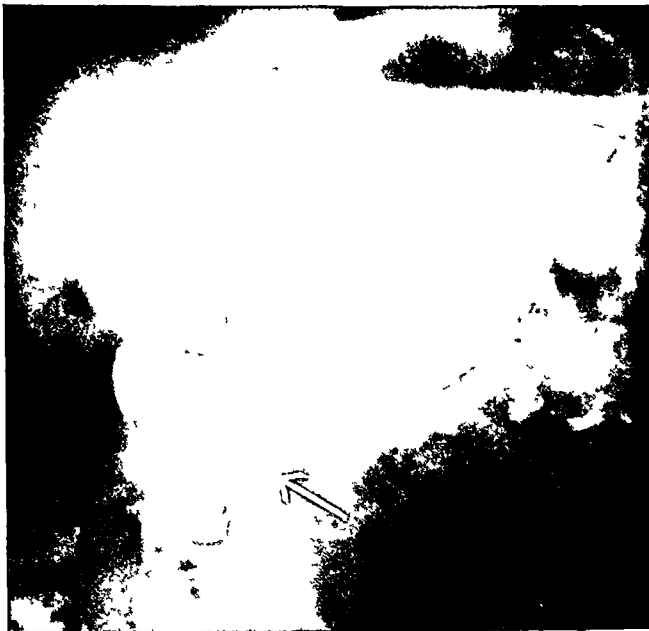


FIG 132 Functional spasm of the stomach Note the narrowing of the pyloric portion However, the mucosal folds are intact

dence of any organic lesion of the stomach or duodenum in spite of very careful examination of this region

The roentgen examination (Fig 132) revealed a persistent narrowing of the pyloric portion of the stomach. The mucosal folds were intact. In the absence of any organic pathology that could be discovered at operation, this narrowing must be considered as functional. The most important evidence as to the functional nature of the pyloric narrowing is the persistence of rugal folds and the absence of any definite irregularity of contour.

THE GASTRIC MUCOSA

The appearance of the rugal folds of the stomach plays an interesting role in clinical roentgenology of this organ. These folds were generally considered to be due to a passive process resulting from the contractions of the muscular coat. Forssell¹ showed that these folds had an independent mechanism, that they varied in appearance from time to time because of active movements of the membrane itself throughout the entire alimentary canal, and that these alterations were conditioned by contractions of the muscularis mucosae.

Thus, according to the work of Forssell, the mucous membrane of the stomach is not a constant anatomic structure but is susceptible of considerable change of an intrinsic nature. Nevertheless, in different divisions of the digestive tract, and in various animals and perhaps in different individuals, there are certain recurring local formations. The lesser curvature of the stomach is more static (Waldeyer's Magenstrasse), whereas the rest of the gastric relief changes considerably. The mucous membrane takes care of the fine regulation of chemical activity, while the musculature is the coarse motor mechanism of the stomach. Forssell believed that an important factor in the changeability of the mucosal relief is the hydrodynamic action of the fluid content of the submucosa which, by its variability, causes corresponding alterations in the thickness of the gastric folds.

The regulatory mechanism probably lies in the plexus of Meissner and in the ganglia of the muscularis mucosae.

Gunn and Underhill (1915), and King and Arnold (1922), published observations on active movements of the digestive mucous membrane of the cat and the dog and Thorell (1924) showed that the surviving muscularis mucosae of man and of warm and cold blooded animals has the capacity of making rhythmic movements of its own, and in combination with nervous elements it responds in a completely typical way to different stimuli, such as physostigmine, barium chloride, papaverine and nicotine.⁴³

Roentgen Aspects. A study of the roentgen appearance of the gastric mucosa is not new indeed it is as old as the history of gastro intestinal roentgenology itself. In the early work of Rieder⁴⁴ one may note roentgenologic evidence of the folds of the stomach. It was Holzknecht,⁴ however, who first emphasized the importance of a routine investigation of the gastric mucosa and recommended palpatory manipulation of the stomach immediately after the ingestion of a small amount of a thin watery suspension of bismuth, in order to accomplish this purpose. Following this preliminary examination, the stomach was completely filled, and a contour study was made.

Special methods for the more detailed study of the mucosal relief were devised by von Elischer,⁴⁵ Lisler and Lenk,⁴⁷ Chaoul⁴⁸ and Berg.⁴⁹

Rendich,⁵⁰ working in the laboratory of the Roentgen Department of Bellevue Hospital, made one of the pioneer contributions to the study of gastric rugae. He emphasized the value of such studies for the more nearly accurate and detailed visualization of ulcer and the radiating folds which may accompany it, for the study of the changes produced by malignancy, for better visualization of the enterostomized stomach and in the study of gastritis. In one normal case two roentgen exposures at an interval

in benign lesions and may be due in part to actual organic involvement in the malignant process (3) Large areas of spastic involvement of the stomach may be noted, particularly in the pars pylorica This region may show marked constriction under fluoroscopic observation with, at times, complete obliteration of the entire region When filled with barium this region is smooth in contour without evidence of destruction of the normal mucosal markings This helps in differentiating the condition from malignant infiltration Moreover, the contour may show variation of outline at different times The administration of atropine to physiologic effect may possibly aid in differential diagnosis by diminishing the spastic manifestations Marked pylorospasm may not necessarily be altogether functional and in some cases it may be associated with the presence of inflammatory disease of the pylorus or may be due to the actual presence of an ulcer (4) In total gastropasm, the entire stomach partakes in the process, and the appearance

may simulate that of a scirrhous carcinoma of the stomach The differential diagnosis lies primarily in the inconstant manifestations in spasm as opposed to the permanent character of the deformity in malignancy, as well as in the demonstration of the intact mucosal relief when the abnormality is spastic in nature Cases of total gastropasm have been described as a result of lead colic, nicotine poisoning and arteriosclerosis of the abdominal vessels

Pylorospasm resulting in marked delay in gastric emptying may be an allergic response to food sensitivity⁴¹

Illustrative Case The alterations in the contour of the stomach produced by functional gastropasm are demonstrated by the following case

M L, male, aged 38 This patient complained of vague gastric discomfort after meals He vomited occasionally. He said that he had lost 40 pounds in the last 5 years He had undergone appendectomy 27 years previously. Physical examination of the abdomen was essentially negative

Exploratory laparotomy revealed no evi-



FIG. 132 Functional spasm of the stomach Note the narrowing of the pyloric portion However, the mucosal folds are intact

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of 2 minutes showed an identical configuration of the mucosal pattern

Technic The following procedure is recommended for the study of the gastric mucosa. For a detailed study of the mucosa to be made by the roentgen method which will approach as nearly as possible actual anatomic conditions, it is essential that examination be conducted on an empty stomach. Unfortunately, such an ideal condition is rarely ever possible, and a small amount of mucus is present under normal conditions which cannot always be removed even by gastric lavage.

A suspension of approximately equal parts of barium sulfate and water is employed, to which a small amount of acacia may be added in order to make the opaque substance stick more intimately to the mucous membrane.

Examination may be conducted with the patient in the erect or in the supine position. The advantage of the supine position is that the suspension is less apt to accumulate at the lower pole of the stomach.

The patient is instructed to drink the mixture, one mouthful at a time. The material is spread manually over the mucosal surface. The additional amount of barium which may be necessary for the patient to swallow will depend upon the individual case. Frequently, a surprisingly small amount of the mixture is sufficient. It is rarely ever necessary to administer more than a total of 150 cc.

Following the distribution of the barium over the mucosal surface in the supine position, the tilt-table may gradually be turned downward until that degree of inclination is obtained which permits the most nearly uniform distribution of the suspension. Varying degrees of compression may be essential in order to bring the mucosal appearance into clearer focus and, by means of a spot-film device, prompt roentgen exposure is made.

Another way in which the gastric mucosa may be demonstrated is for the stomach to be filled with a larger amount of barium,

sufficient for contour study. The patient is placed in the prone position and turned slightly to his right in order to avoid excessive pressure against the stomach. Films are taken at varying intervals as the stomach gradually empties itself of the barium suspension. These films are developed promptly, and further observations are made as required by the individual case. An examination conducted in this manner will yield valuable information regarding the roentgen appearance of the gastric mucosa and of the external configuration of the stomach itself.

A word of caution is necessary. In a desire to study the mucosal relief in all its detail, one must not lose sight of the fact that this is only an additional procedure which under no circumstances is to supplant the more fundamental method of studying the stomach by completely filling it. The most valuable information in the examination of the stomach is still to be obtained by this procedure, and mucosal studies must be considered only as an accessory although highly important aid.

A number of factors influence the roentgen appearance of the mucosal relief.

Mucus present in the stomach may influence the gastric relief in two ways: (1) by producing a negative shadow within the barium relief, and (2) by becoming impregnated with barium similar to the process which has been shown to occur in the roentgen examination of the colon in the presence of mucus. An exaggeration in the production of mucus may increase this tendency. Moreover, if the excess mucus forms a coating on the gastric mucous membrane, there may be an absence or an apparent irregularity of the folds not due to any intrinsic derangement of the anatomic characteristics of the mucosa itself.

Occasionally, small globules of air may be mixed with the gastric secretion and produce translucent areas in the gastric relief.

The differentiation of translucent areas produced by mucus and air from actual intrinsic changes in the mucosa may be

made in two ways (1) by the fact that such translucent areas may be moved about by palpatory manipulation behind the fluoroscopic screen and (2) because re-examination, particularly after preliminary gastric lavage may fail to show the recurrence of this phenomenon. Similarly, the presence of food in the stomach either because of improper preparation of the patient or because of retention due to pyloric stenosis may also produce translucent areas of varying sizes in the roentgenogram. Similarly these may be differentiated from intrinsic gastric pathology by inconstancy of localization under manipulation and as the result of re-examination.

In addition to those factors which have already been considered such as retained gastric secretion, food, mucus and globules of air, the appearance of the mucosal relief picture may also be influenced by the thinness of the contrast mixture employed and the uniformity of its distribution. Obviously the thicker the mixture employed, the greater the opportunity of covering the rugal folds in addition to the grooves between them, so that fewer folds of an apparently distorted character may appear in the roentgenogram. Similarly lack of homogeneity in the distribution of the barium may be responsible for apparently bizarre relief pictures. Thus if barium fails to enter a groove the translucent areas in the roentgenogram may represent not only the actual fold itself but also the width of the barium free groove in addition. The quantity of barium suspension employed may also influence the apparent size of the folds since dilatation of the stomach may produce various degrees of obliteration.

Another important factor is the degree of mechanical pressure which is exerted upon the stomach. Soft, readily pliable folds may be completely obliterated by excessive pressure. Areas free of folds may be caused thereby in the neighborhood of a peptic ulcer which may be falsely interpreted as being due to the displacement of the barium by abnormal swelling of the

folds. Insufficient pressure may either fail to show the folds in a clear manner or may give the impression of haziness of outline. It is also possible that improperly applied pressure may cause an artificial appearance of puckering in the roentgenogram.

Still another factor capable of producing a bizarre roentgenogram is that which comes from the overlapping of anteriorly and posteriorly placed folds. In the normal mucosal relief folds may be noted which are longitudinal in direction, hugging the lesser curvature at the Magenstrasse. Occasionally, a longitudinal fold of the esophagus may be noted continuous with the fold of the lesser curvature. The rest of the folds of the stomach run fairly parallel as far as the region of the incisura angularis, when they diverge in a somewhat spiral manner toward the distal portion of the greater curvature. Occasionally the folds lack this divergence and run through the entire length of the pylorus in longitudinal fashion. In the immediate prepyloric region the folds may occasionally run in a transverse direction. There may be a convergence of folds at the entrance to the pyloric ring. Along the greater curvature the folds are more tortuous and frequently show characteristic toothlike serrations. The folds and the grooves between them may show considerable variation within normal limits.

Mucosal relief studies of the pars cardiae may show a small rounded translucent area high up near the lesser curvature, produced by the entrance of the esophagus. This appearance must not be confused with a tumor. Differential diagnosis depends on (1) the fact that it corresponds anatomically to the position of the entrance of the esophagus, (2) verification of this fact when there is any doubt by combined examination of the barium filled esophagus and stomach, and (3) changes in the size of the translucent area or its complete absence at times, depending on transient variations in the degree of dilatation of the cardiac end of the esophagus where it enters the stomach.



FIG 133 (*Top*) Normal mucosal folds of the stomach



FIG 134 Mucosal folds in the pars cardia. Note the translucent area produced by the entrance of the esophagus



FIG 135 Normal mucosal folds of the pars media of the stomach

Illustrative Cases The appearance of the normal gastric mucosa is shown in the following roentgenograms

Figure 133 shows mucosal folds throughout the stomach running, for the most part, in an essentially parallel manner. There is also evidence of decussation of some of the mucosal folds.

A normal pattern of the mucosal folds in the pars cardia is illustrated by Figure 134. Note the irregularly rounded, translucent area high up, produced by the entrance of the temporarily distended esophagus.

Figure 135 shows the vertical mucosal folds of the stomach in the pars media. These folds run essentially parallel with one



FIG. 136 Normal mucosal folds of the pars media



FIG. 137 Mucosal folds of the pars pylorica



Fig. 138. Normal mucosal folds of the pars pylorica



Fig. 139. Rounded translucent areas produced by mucosal folds in cross section



another. They exhibit a moderate degree of tortuosity.

Figure 136 shows the mucosal folds at the junction of the pars pylorica and the pars media, running in a manner essentially parallel with one another. A moderate degree of tortuosity may be noted, particularly near the greater curvature border.

Figure 137 shows the mucosal folds in the pyloric portion of the stomach. In this area the tendency may be noted for the mucosal folds to be directed from the lesser curvature toward the distal portion of the opposite greater curvature.

The folds in the pars pylorus may at times run parallel. Small, rounded translucencies in the course of the folds apparently represent bits of mucus or tiny air bubbles (Fig. 138).

Some of the larger, more clearly defined rounded translucent areas represent mucosal folds in cross section (Fig. 139). They may be confused with small polyps or abnormal polypoid changes. Permanently present when the cause is actual organic disease, the pattern will change in repeated observations of the identical region when

Fig. 140. Prominent and tortuous mucosal folds of the stomach

the condition is caused by normal mucosal folds

At times, the folds may be unusually prominent and tortuous (Fig 140) and may be, nevertheless, the result of functional changes in the absence of an actual gastritis. The suspicion of underlying inflammatory pathology as the cause is much stronger when the mucosal pattern shows the very marked changes to be seen in Figure 141. The relation of such findings to the diagnosis of chronic gastritis will be discussed in the next chapter.

REFERENCES

- Hesse, A. Geben uns die in der Radiologie zur Verwendung kommenden Metallsalze ein falsches Bild von Form und Grösse des Magens? Berlin klin Wchnschr 48 931 1911
- Groedel, F., and Seyberth, L. Tierexperimentelle Untersuchungen über den Einfluss der Röntgenmahlzeit auf die Magenform, Arch Verdauungskr 18 8 1912
- Rieder, H. Radiologische Untersuchungen des Magens und Darmes beim lebenden Menschen. München med Wchnschr 51 1548, 1904
- Holzknacht, G. Zur Röntgendiagnose der Magenatonie, Wien med Wchnschr 6 1046 1912
- Forssell, G. Über die Beziehungen der Röntgenbilder des menschlichen Magens zu seinem anatomischen Bau, Fortschr Geb Röntgenstrahlen supp 30, 1913
- Kastle, C., Rieder, H. and Rosenthal, L. Über kinematographische aufgenommene Röntgenogramme (Bio Röntgenographie) der inneren Organe des Menschen, München med Wchnschr 56 280 1909
- Kaufmann, R. and Kienbock, R. Über den Rhythmus der Antrumperistaltik des Magens, München med Wchnschr 58 1237, 1911
- Lewis, Frederick T. Forms of the stomach in human embryos with notes upon the nomenclature of the stomach. Am J Anat 13 477, 1912
- Lisek, G. R. and LeWald, L. T. The further study of the anatomy and physiology of the infant stomach based on serial roentgenograms. Am J Dis Child 6 232, 1913
- Bouslog, J. S., Cunningham, R. D., Haner, J. P., Walton, J. B. and Waltz, H. D. Roentgenologic studies of the infant's gastrointestinal tract. J Pediat 6 234 1935
- Schlesinger, E. Die Grundformen des normalen und pathologischen Magens und ihre Entstehung, Berlin klin Wchnschr 47 1977, 1910
- Klee, P. Die Magenform bei gesteigertem Vagus und Sympathikustonus. München med Wchnschr 61 1044 1914
- Mills, R. W. The relation of bodily habitus to visceral form, position, tonus and motility, Am J Roentgenol 4 155 1917
- Spiegel, Bela. Einklemmung des Magens im Schenkelbruch. Zentralbl Chir 47 373 1920
- Ahrens, Philipp. Einklemmung des Magens im Schenkelbruch. Zentralbl Chir 47 1345, 1920
- Stokes, A. F. Strangulated right inguinal hernia containing stomach and transverse colon. M J Australia 9 187 1922
- Siegmund, H. Die erworbenen Lage und Gestaltsabweichungen des Darmtrunkes



FIG 141. Marked enlargement and distortion of the mucosal folds of the stomach.

- Handbuch der Speziellen Pathologischen Anatomie und Histologie, vol 4, part III, p 129, Berlin, Springer, 1929
- 18 Lust, F J Herniation of the stomach into the scrotum Report of case, *Am J Roentgenol* 37 666, 1937
 - 19 Hartley, J B Diverticulum of stomach found to enter left inguinal hernial sac, *Brit J Radiol* 18 231, 1945
 - 20 Cave, Paul Stomach in a femoral hernia, *Brit J Radiol* 21 143, 1948
 - 21 Ladd, W E, and Gross, R E *Abdominal Surgery of Infancy and Childhood*, p 85, Fig 53, Philadelphia, Saunders, 1941
 - 22 McCutchen, G T Reduplication of the stomach Report of a case, *Ann Surg* 129 826, 1949
 - 23 Miller, J M, and Ginsberg, M Congenital duplication of stomach, *Arch Surg* 60 995, 1950
 - 24 Gairdner, W T A remarkable cyst in the omentum, *Tr Path Soc London* 3 374, 1851-1852
 - 25 Bantock, G G Cyst of the great omentum, *Tr Obst Soc London* 23 164, 1881.
 - 26 Hasbrouck, E M Enormous endotheliomatous cyst of the great omentum, *Ann Surg* 48 206, 1908
 - 27 Dowd, C N Cysts of omentum, *Ann Surg* 54 617, 1911
 - 28 Pybus, F C A case of large omental cyst in a child, *Lancet* 1 63, 1917
 - 29 Horgan, J Cysts of the omentum a review and report of a case, *Am J Surg* 29 343, 1935
 - 30 Birnkrant, Milton Traumatic serous cyst of the lesser omentum, *Radiology* 42 74, 1944
 - 31 Kampmeier, R H Aneurysm of the abdominal aorta a study of 73 cases, *Am J M Sc* 192 97, 1936
 - 32 Hubeny, M J, and Pollack, Simon Saccular abdominal aortic aneurysm, *Am J Roentgenol* 43 385, 1940
 - 33 Eliason, E L, and McNamee, H G Abdominal aneurysm—a report of 24 cases, *Am J Surg* 56 590, 1942
 - 34 Scott, V Abdominal aneurysms, *Am J Syph, Gonorr & Ven Dis* 28 682, 1944
 - 35 Pratt-Thomas, H R Aneurysm of abdominal aorta—analysis of 17 cases, *J South Carolina M A* 40 251, 1944
 - 36 Engel, A, and Lysholm, E A new roentgenological method of pancreas examination and its practical results, *Acta radiol* 15 635, 1934
 - 37 Leven, G L'aerophagie, syndromes gastriques, intestinaux, circulatoires et respiratoires, ed 2, Paris, Doin, 1926
 - 38 Jollasse, O Zur Motilitätsprüfung des Magens durch Röntgenstrahlen, *Fortschr Geb Röntgenstrahlen* 11 47, 1907
 - 39 Haudek, M Die Technik und Bedeutung der radiologischen Motilitätsprüfung, *Verhandl deutsch Gesellsch inn Med* 29 143, 1912
 - 40 Holzknecht, G, and Luger, A Zur Pathologie und Diagnostik des Gastrosasmus, *Mitt Grenzgeb Med u Chir* 26 669, 1913
 - 41 Fries, Joseph H Roentgen studies of allergic children with disturbances of the pylorus resulting from food sensitivity, *J Allergy* 23 39, 1952
 - 42 Forssell, G Studies of the mechanism of movement of the mucous membrane of the digestive tract, *Am J Roentgenol* 10 87, 1923
 - 43 Quoted by Forssell, G The motor mechanism of the digestive mucous membrane, *Brit J Radiol* 31 189, 1926
 - 44 Rieder, H Beiträge zur Topographie des Magen-darmkanales beim lebenden Menschen nebst Untersuchungen über den zeitlichen Ablauf der Verdauung, *Fortschr Geb Röntgenstrahlen* 8 147, 1905
 - 45 Holzknecht, G Die neueren Fortschritte der Röntgenuntersuchung des Verdauungstraktes, *Berlin klin Wchnschr* 48 158, 1911
 - 46 von Elischer, J Über eine Methode zur Röntgenuntersuchung des Magens *Fortschr Geb Röntgenstrahlen* 18 332, 1912
 - 47 Eisler, F, and Lenk, R Die Bedeutung der Faltenzeichnung des Magens für die Diagnose des Ulcus Ventriculi, *Deutsche med Wchnschr* 47 1459, 1921
 - 48 Chaoul, H Systematische Röntgenuntersuchung am Schleimhautbild des Magens und Duodenum Darstellbarkeit und Verwertbarkeit, *Acta radiol, supp III, pt II*, p 26, 1929
 - 49 Berg, H H Röntgenuntersuchung am Innenrelief des Verdauungskanales Ein Beitrag zur klinischen Röntgendiagnostik insbesondere von Entzündung, Geschwür und Krebs, Leipzig, Thieme, 1930
 - 50 Rendich, R A The roentgenographic study of the mucosa in normal and pathological states, *Am J Roentgenol* 10 526, 1923

Chronic Gastritis

ETIOLOGY AND PATHOLOGY

One of the earliest descriptions of an erosive gastritis was that by Morgagni¹ in Epistle 29, No 20, of his famous work. The stomach was full of erosions, some of which had become gangrenous. Some of the erosions were close together and were very small. In addition to the gastric erosions, he also found erosions in the proximal portion of the duodenum and in the esophagus. He believed that the condition was probably due to something the patient had eaten.

In Figures 1 and 2 (Plate 2, Installment 30) Cruveilhier showed beautiful examples of what he described as a follicular gastritis. In the duodenum and throughout the small intestine scars were present similar in nature to those in the stomach. Cruveilhier stated that follicular gastritis had not received the clinical recognition the condition deserved, although such ulcerative inflammation was a common form of gastritis. He described cases in which the entire mucosa was involved in superficial erosions. Hemorrhagic erosions of the stomach might be present causing hematemesis. He described the appearance in a number of cases and included excellent reproductions of the autopsy findings.

Rokitansky³ recognized the existence of a gastritis mucosa associated with hypertrophy of the mucous membrane, which in an advanced degree might show a warty surface, leading to the formation of polyps. He found chronic catarrhal inflammation to be most common in the pyloric portion of the stomach.

The existence of chronic gastritis was also described in the works of Brinton⁴ and Dieulafoy.

The serious difficulty in the study of the pathology of chronic gastritis was due to the changes which developed postmortem. A great advance in the study of the problem resulted from the contribution of surgery, which yielded fresh material obtained by partial gastrectomy.

The changes produced by chronic gastritis may be seen in the fresh specimen, and the characteristics may be retained if the specimen while still warm, is fixed in formalin. The gastric mucosa is irregular in appearance. It may show polypoid proliferations and the presence of acute inflammation with fresh or healed erosions. In advanced cases, the deeper tissues of the wall of the stomach may be involved with rigidity and hypertrophy and occasionally with narrowing of the pyloric antrum. The serosa may show reddening with the production of adhesions and inflammatory hyperplasia in neighboring lymph nodes.

ROENTGEN DIAGNOSIS

The evidence obtained on roentgen study of the gastric mucosa must be interpreted with considerable care before one can safely conclude that it necessarily justifies a diagnosis of intrinsic organic disease. This conservative attitude is based upon the following observations:

1. Increased prominence and tortuosity of the mucosal folds may in some cases be due to a reflex stimulation of the gastric innervation.⁶ The vegetative nervous sys-

tem apparently plays a great role, influencing the behavior of the mucosal relief through its effect upon the muscularis mucosae. Therefore, the significance of changes in the gastric relief must be interpreted in the light of the functional behavior of the patient from the standpoint of autonomic balance.

2 Changes in the mucosal relief with increased prominence of the gastric folds may be the expression of an allergic response to food⁷ and therefore may be the result of a functional derangement and not of organic disease.

3 Abnormal prominence of the mucosal folds may be due to the presence of giant rugae of congenital origin.⁸ In such cases a localized, circumscribed thickening of the mucosal relief may have no bearing whatever as an indication of an actual gastritis. Prominent rugae of this nature may show a pure hyperplasia of the mucosa without any evidence of inflammation.

4 Particularly significant is the fact that there is no definite correlation between the nature of the folds, as seen roentgenologically, and their actual appearance as observed gastroscopically. Only in those cases in which translucent areas in the roentgenogram correspond to nodular wartlike structures in the mucosa is the roentgenographic evidence acceptable.

Unusually prominent mucosal folds may be present in the absence of any evidence of organic derangement. On the other hand, in some cases in which the roentgen manifestations appear to be those of a normal mucosal relief, definite evidence of organic change of the mucosa has been found. Thus, in some cases of pernicious anemia with atrophic gastritis, roentgen examination may show the presence of apparently normal gastric rugae. This appears to be due to the ability of the muscularis mucosae to throw even an atrophic mucosa into folds. Narrowing of the mucosal folds, therefore, is not to be considered an evidence of an atrophic gastritis but is rather to be attributed to a diminution of the tone and

turgor of the gastric submucosa which is associated with various degrees of dehydration.

5 Still another difficulty in the roentgen diagnosis of chronic gastritis lies in the fact that, even with a painstaking technic, it is not always possible to obtain an exact anatomic representation of the gastric rugae. Thus mucus, which it may be impossible to remove completely, may adhere to the grooves between folds, thereby preventing the entrance of barium. Under such circumstances, folds in the roentgenogram may appear deceptively wide. Moreover, a slight increase in the thickness of the barium suspension, or the application of an improper degree of compression, or the overlapping of anteriorly and posteriorly placed folds, may add to the difficulties and distort the picture. In addition, adherent flecks of mucus may displace the barium, with the production of translucent areas which may mimic those produced by actual excrescences of the mucosa of a pathologic nature. These technical difficulties are therefore added reasons for a conservative approach to the diagnosis of chronic gastritis on the basis of the roentgen data alone.

It cannot be emphasized too strongly, therefore, that the roentgen diagnosis of gastritis is subject to considerable error of interpretation. To base the diagnosis of gastritis upon apparent increased thickness and tortuosity of the rugal folds as they appear in the roentgenogram is not justified, since they may be functional in nature or, in rare instances, congenital in origin and not necessarily the expression of organic disease. Gastroscopy in particular has called our attention to the danger of reading the presence of organic disease into a gastric relief of this nature. Just as prominence of the mucosal folds in the roentgen relief does not necessarily justify the diagnosis of a hypertrophic gastritis, so an apparent diminution in the size of the folds does not justify the diagnosis of an atrophic gastritis. Moreover, definite pathologic evidence of gastritis is not incompatible with

an apparently normal size and distribution of the mucosal folds in the roentgen relief

Since gastritis is a comparatively frequent disease, it is obvious that the roentgen diagnosis of chronic gastritis will often appear to be corroborated, though this may be simply due to the law of chance. In the diagnosis of chronic gastritis, gastroscopic study is apparently far more reliable in general than the most painstaking and careful roentgen technic.

Are there any roentgen features which may justify the assumption that an actual chronic gastritis is present? The most reliable finding is that of small, fairly well rounded translucent areas of a permanent character, representing actual wartlike excrescences or polypoid changes. Here too, however, one must be certain that such translucencies are really permanent, since clumps of mucus or small particles of retained food may produce similar changes. However, such alterations are of a temporary character, and palpatory manipulation under fluoroscopic control, as well as re-examination, should demonstrate their evanescent nature.

Spriggs and Marver⁹ described 19 cases of polypoid hyperplastic swelling of the gastric mucosa, with correlation of the roentgen changes and the anatomic appearance of the lesion.

While unusually prominent and tortuous mucosal folds are not necessarily the result of a pathologic process, extreme exaggeration of these folds associated with a lack of pliability may furnish evidence of a suggestive nature. This is illustrated by the well authenticated case reported by Cole.¹⁰

At times the infiltrative character of the hypertrophic gastritis may produce changes in the roentgen appearance of the stomach indistinguishable from malignant infiltration. The occasional difficulty in differentiating between the deformity produced by chronic gastritis and carcinoma is illustrated by the carefully controlled studies of Freedman, Glenn and Laipply¹¹ and the well documented report by Hinkel.¹² In

Hinkel's case both the roentgen evidence of deformity of the pars cardiae and the gross appearance of the resected specimen (photographs of which are included in the report) were certainly indicative of an intramural tumor. Microscopic examination showed an adenomatoid hyperplasia of the mucous glands with chronic diffuse gastritis.

The mimicry of malignant infiltration in the roentgenogram of the stomach produced by giant hypertrophic gastritis has been amply confirmed.^{13, 14}

On one of the four divisions (Cornell) at Bellevue Hospital three cases were encountered in a 10 year period in which not only the preoperative roentgen evidence but also the gross deformity at operation simulated gastric carcinoma. That the lesions were due to a hypertrophic gastritis was established only on the basis of microscopic examination.¹⁵ Cases of a similar nature have also been encountered on all of the three other divisions of the hospital.

In a desire to correlate the roentgen findings in the mucosal relief with evidence of organic change, one must not lose sight of the fact that alterations of contour in the completely filled stomach may also be noted in the presence of chronic gastritis. The irregularity of contour may be demonstrable in two ways: (1) Both the lesser and greater curvatures may appear extremely jagged. The fact that the lesser curvature is involved is of great significance, since the greater curvature itself may often exhibit some degree of irregularity due to the normal distribution of the mucosal folds in this region. (2) Various degrees of irregular narrowing, not of a jagged character, may distort the contour particularly in the antral region. These changes may be associated with various degrees of narrowing and stenosis of such nature as actually to make hazardous or impossible differentiation from malignant infiltration. Superadded spasm may further distort the picture and exaggerate the difficulties of differential diagnosis.

plaint of cough, fever and chill of 3 days' duration. He had had some epigastric pain for the past 2 weeks. A diagnosis of pneumonia involving the left lobe was made at that time. About 2 weeks after admission, he vomited blood following an attack of severe epigastric pain. He then stated that he had had several similar attacks 2 years previously.

Physical examination revealed the abdomen to be tender, and spasm was noted in the midepigastric region. The Wasserman reaction was negative.

Fractional gastric analysis revealed complete absence of free hydrochloric acid.

The operative findings were: "The stomach is of average size. There are no gross masses. The wall of the stomach feels more firm and thickened than usual, especially at the distal end in the prepyloric region. There are several soft but definitely enlarged lymph nodes in the mesentery and greater omentum. The liver is normal in size and consistency. No palpable masses are present in the liver. Resection of the distal third of the stomach was done."

Pathologic examination of the resected stomach revealed the following: "The specimen consists of the distal third of the stomach. The mucosa is intact and the wall feels thicker than normal. However, no evidence of malignancy is noted. Microscopic examination of the section reveals thickened mucosa and some fibrosis of the wall with some round-cell in-

vasion. The glands appear normal. There is no invasion beneath the muscularis mucosa. No evidence of malignancy is present."

Examination of a lymph node of the mesentery revealed the following: "The general architecture is fairly well preserved, but there is hyperplasia of the reticulo-endothelial cells. The germinal centers share in this hyperplasia to a slight extent. Some of the sinuses are dilated and contain reticulo-endothelial cells. There is no evidence of metastatic malignancy."

The diagnosis was reticulo-endothelial hyperplasia of the lymph nodes.

Roentgen examination (Fig 143) revealed a persistent narrowing of the distal portion of the stomach. The degree of narrowing was most marked in the immediate prepyloric region. There was a smooth broad defect of the greater curvature pars media with a superficial irregularity of the greater curvature distal to this area. The pyloric ring was normal. The duodenal bulb was elongated but was otherwise normal. The preoperative diagnosis was an infiltrative lesion involving the distal portion of the stomach, the most likely cause being thought to be scirrhus carcinoma. As determined by both macroscopic and microscopic study the cause of this deformity was a chronic hyperplastic gastritis.

The mimicry of carcinoma by a marked hypertrophic gastritis is illustrated here.



FIG 143 Chronic gastritis

N P male aged 60 The patient gave a 7 year history of recurrent attacks of non radiating epigastric pain gradually increasing in severity The pain might occur at any time and occasionally it woke him from sleep It was relieved by the ingestion of food particularly milk and cream Tarry stools were noted on occasion He lost from 10 to 15 pounds during his illness Physical examination was essentially negative

Radiographic examination (Fig. 144) showed a marked persistent narrowing of the distal half of the stomach with irregularity of contour and a niche on the lesser curvature The preoperative diagnosis based on these findings was that the lesion was malignant Exploration revealed an ulcer on the posterior wall of the stomach The pyloric antrum was thickened The posterior wall of the stomach was adherent to the transverse mesocolon at the site of the ulcer No other pathology was noted A subtotal gastrectomy was done

Macroscopic Examination "Upon opening the stomach hypertrophic rugae are seen In the pars media on the posterior aspect is a large indurated ulcer with thick edges which extends to the serosal surface There is an other small, indurated ulcer in the pars pylorus lesser curvature There are several small firm nodes in the gastrohepatic ligament On section the ulcers have penetrated through all layers"

In the *microscopic examination* it was noted that the mucosa was thickly infiltrated with lymphocytes and plasma cells

Diagnosis Chronic ulcers of stomach Chronic gastritis

The *pathologic examination* therefore revealed that the markedly thickened and hypertrophic rugae which distorted the configuration of the stomach were the result of a chronic gastritis associated with ulcerations

The next case is an example of the marked degree of deformity of the stomach which may be produced after swallowing lye simulating the deformity of a malignant lesion

C T male aged 20 Fifteen days before admission to the hospital, the patient awoke in the morning with a nosebleed He drank a glass of what he thought was water and immediately thereafter he had a sharp burning pain in his throat and stomach, following which he vomited blood He had difficulty in swallowing thereafter and 5 days later vomited



FIG 144 Chronic gastritis with ulceration Note the narrowing and the irregularity of contour of the distal portion of the stomach and the niche on the lesser curvature The preoperative roentgen diagnosis was carcinoma

an esophageal cast (proved microscopically) Visualization of the larynx and the esophagus showed ulceration throughout It was later learned that the fluid which the patient had imbibed was lye

Esophagoscopy later revealed numerous ulcerations combined with esophagitis Five days after his admission to the hospital, the patient vomited practically all his solid and liquid food

At operation marked thickening was found in the pyloric region, this had apparently produced a partially obstructing lesion A biopsy was taken from the stomach wall

The microscopic examination showed the mucosal surface to be covered with an exudate of clotted blood cellular debris and polymorphonuclear and mononuclear cells The mucosa itself was eroded in many places The glands showed a uniform granular degeneration in many places reaching complete disorganization although the bases of the glands were well preserved

A gastro enterostomy was done as well as a gastrotomy

Roentgenographic examination about 1 month after the operation revealed the following

- 1 An extreme narrowing of practically the



FIG 145 A Narrowing and obstruction of the distal portion of the stomach due to the swallowing of lye

entire esophagus of the type seen as a result of chemical corrosion

2 Examination of the stomach (Fig 145 A) showed practically complete occlusion of the pylorus with a funnel-shaped appearance of the stomach proximal to the area of constriction. There was a gastro-enterostomy with only a small amount of barium leaving the stomach. The gastrostomy tube was also noted. The appearance of the stomach was not at all that usually seen as a result of an infiltrative lesion. The history, the findings at operation and the pathologic examination of the biopsy specimen all showed that the pyloric occlusion was the result of a severe gastritis due to lye.

Another example of pyloric deformity resulting from corrosive gastritis due to the effect of the ingestion of lye may be noted in the following case.

J. T., male, aged 40. As the result of the swallowing of lye the patient was admitted to the hospital with severe burns about the mouth, the pharynx and the esophagus. The patient's esophagus was dilated daily, and esophagoscopy 6 weeks later was reported as showing normal mucosa without stricture. A few days thereafter he complained of abdominal pain and frequent vomiting.



FIG 145 B Annular deformity of the pylorus of inflammatory origin in a patient who swallowed lye

Roentgenographic examination (Fig 145 B) revealed evidence of an annular constriction of the pylorus with a pouchlike dilatation of the lesser curvature. There was considerable obstruction produced by this lesion. At 5 hours about three fourths of the barium was still present in the stomach. The conclusion was pyloric obstruction of organic origin, in all probability secondary to the effect of the swallowed lye. The evidence was also strongly suggestive of an ulcerating lesion within the major inflammatory process.

At operation there was an annular type of constricting prepyloric lesion. A subtotal gastrectomy was done. "The pylorus is enlarged and hardened measuring approximately 4 cm in diameter and 3 cm in length. The wall of the pylorus is found to be markedly thickened. The mucosa of the pylorus and that of the stomach just proximal to the pylorus for a distance of about 2 cm is superficially ulcerated and edematous."

Based on the microscopic examination, the final pathologic diagnosis was Chronic gastritis. Fibrosis of the pylorus and prepyloric portion of the stomach.

GRANULOMA OF THE STOMACH WITH EOSINOPHILIC INFILTRATION

An unusual granulomatous lesion of the stomach has been described characterized mainly by infiltration of the wall with eosinophils. It is believed that these organic changes may represent an allergic manifestation of the digestive tract. Kaiser¹⁰ in 1937 described three cases of allergic manifestations in different parts of the alimentary tract.

One patient, a male, age 53, had a family background of allergic manifestations. He, himself, had abdominal pain and vomiting after eating onions. A partial gastrectomy was done for a peptic ulcer. Nine days later examination of the blood showed 25 per cent eosinophils. Pathologic examination of the resected specimen showed edema and swelling of the submucosa. There was widespread infiltration of the mucosa and the submucosa with plasma cells and eosinophils.

Massive infiltration with eosinophils was also described by Barrie and Anderson.¹⁰

The patient, a married female, 27 years old, gave a history of pain in the umbilical region sufficiently severe to keep her awake at night. She was relieved by vomiting. A similar less severe attack had occurred 4 years previously. There was no history of asthma or urticaria. Eggs, bacon and pastry caused indigestion. A roentgenogram of the stomach (which is not included in the article) was reported as showing a smooth filling defect of the greater curvature of the prepyloric portion of the stomach. The mucosal pattern of the involved segment was intact.

There was marked eosinophilia in the blood examination. At operation the pylorus was found to be greatly thickened. In addition the duodenum and the proximal 2 feet of the jejunum were thickened, indurated and friable. After a partial gastrectomy, the increase of the eosinophils in the blood persisted. Histologic study of the resected specimen showed a number of interesting features:

1. Hypertrophy of the muscular coat of the stomach, the pylorus and the duodenum.

2. Massive infiltration of the pylorus with eosinophils.

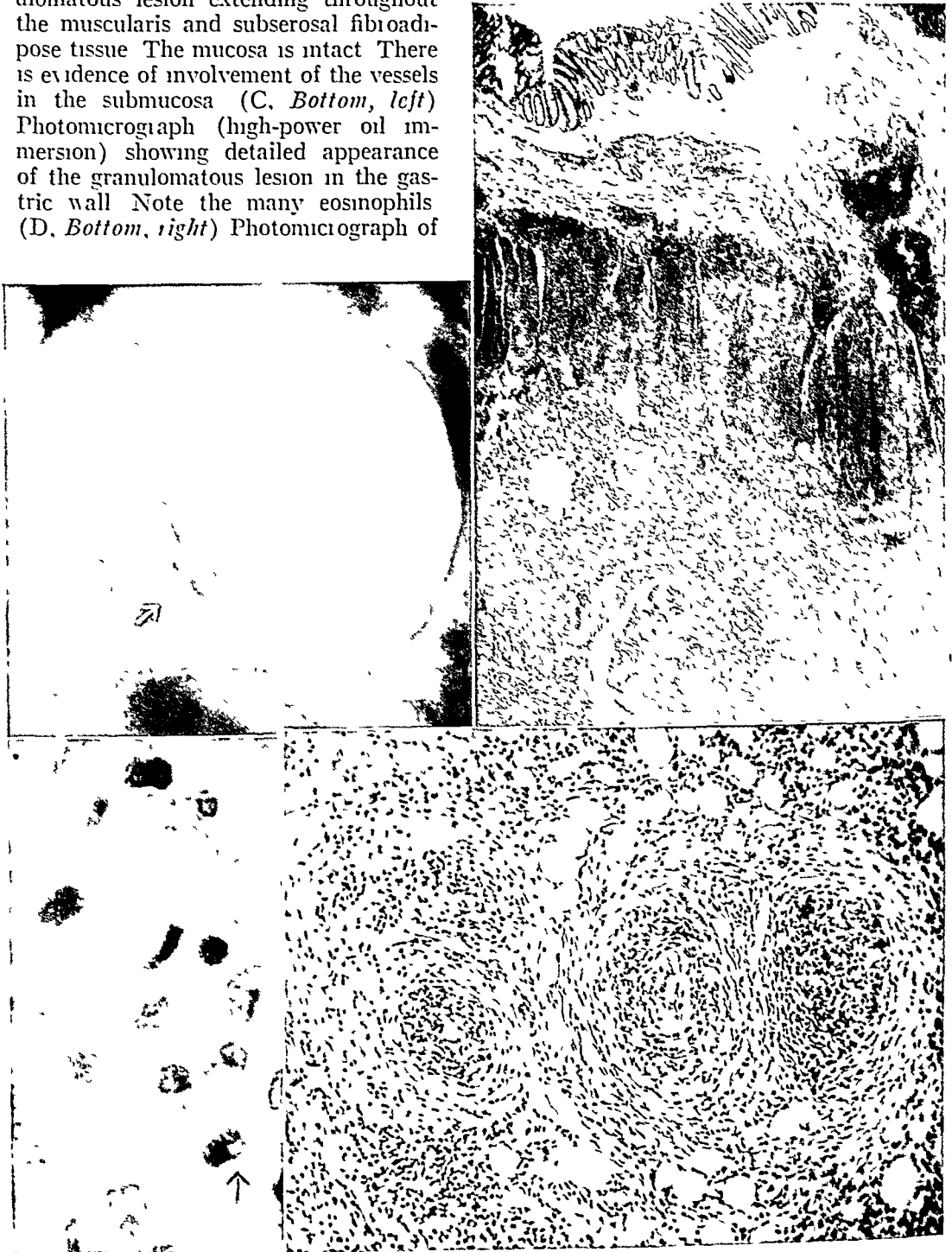
3. Unusual para arterial giant cell follicles.

When eosinophils are found in the intestinal tract in chronic inflammatory lesions, they are most numerous in the mucosa. In the case described by Barrie and Anderson there were very few eosinophils in the mucosa. They were present mainly in broad bands between the muscle fibers. There was an almost complete absence of plasma cells and fibrosis which is usually present in inflammation. They believed that this localized eosinophilic invasion represented an allergic reaction that the eosinophils were not produced locally but originated from the blood stream. They also believed that the peculiar giant cell follicles were possibly evidence of sensitivity. They appeared to be similar to the giant cell follicles described by Sisk¹ in 4 cases of eosinophil myocarditis which developed after neoplasia after treatment of cardiac disease.

The essential feature in the pathology of Vanek's cases was an infiltration of the wall of the stomach with many eosinophils. The roentgen appearance of the deformity of the stomach was not included. The peculiar lesion which characterized his cases

FIG 146 Granuloma of the stomach with eosinophylic infiltration and periarteritis (A, *Top, left*) Note the deformity particularly of the pyloric portion of the stomach produced by the lesion (B, *Top, right*) Section of the wall of the stomach, showing the granulomatous lesion extending throughout the muscularis and subserosal fibroadipose tissue The mucosa is intact There is evidence of involvement of the vessels in the submucosa (C, *Bottom, left*) Photomicrograph (high-power oil immersion) showing detailed appearance of the granulomatous lesion in the gastric wall Note the many eosinophils (D, *Bottom, right*) Photomicrograph of

the vessels in the subserosal adipose tissue There is necrosis of the vessel wall All layers are infiltrated by mononuclear cells and polymorphonuclear cells, of which a fair number are eosinophils



consisted of more or less loose collagenous tissue with fibroblasts lymphocytes and eosinophils. The lesion appeared primarily as a circumscribed focus in the submucosa spreading toward the mucosa of the stomach in some cases even producing polyp formations. The lesion was therefore essentially a gastric submucosal granuloma with eosinophilic infiltration.

Eosinophilic infiltration of the wall of the stomach and the proximal small intestine was also described by Spencer Comfort and Dahlin.²³ The story was that of a physician 40 years of age, who during a period of 12 years had recurring episodes of epigastric and lower abdominal cramps associated with diarrhea and vomiting. His leukocyte count showed an eosinophilia at one time as high as 63 per cent. Roentgen examination revealed a dilated stomach with a small gastric residue at 18 hours but without any evidence of a deformity of the stomach or duodenum to account for this delay. Operation revealed marked thickening of the pyloric ring and of the duodenum. Microscopic examination of the resected specimen showed a diffuse infiltration of the muscularis with eosinophils which were present in sheets between the muscle bundles. A similar condition was found in a biopsy specimen taken from the jejunum.

Additional examples of tumefaction of the stomach associated with eosinophilic infiltration were reported by Doniach and McKeown⁴ and Ruzic, Dorsey and Huber.

I have had one case of a granulomatous lesion of the stomach characterized by eosinophilic infiltration of the wall with periarteritis. The patient (A. S., male, aged 63) stated that during the preceding 6 weeks he had pain across the midabdomen, occurring from 5 to 10 minutes after meals and lasting for several hours. The pain was relieved by bicarbonate of soda. On one occasion the pain woke him from sleep. There was no history of vomiting, hematemesis or blood in the stool. He lost about 5 pounds in the preceding month.

Physical examination of the abdomen was essentially negative. Gastric analysis revealed almost complete absence of hydrochloric acid.

Roentgenographic examination (Fig. 146 A) revealed a tubular narrowing of the pyloric antrum. The greater curvature of the pars pylorus appeared to be rigid and was unaltered by peristaltic activity during prolonged periods of observation on several occasions. An umbilicated type of niche extended from the lesser curvature of the pars pylorus. On the basis of these findings a diagnosis of an infiltrative lesion was made justifying surgical resection.

At operation the distal half of the stomach was markedly thickened and so adherent to the transverse colon that in the resection of the stomach the adherent colon had to be included.

The pathologic findings in this case were extremely interesting. Figure 146 B shows the low power appearance of a section through the granulomatous lesion. The presence of the many eosinophils in the granulomatous lesion of the gastric wall may be noted in Figure 146 C (high power oil immersion). Figure 146 D is a photomicrograph of the vessels in the submucosal adipose tissue. There is necrosis of the vessel wall. All layers are infiltrated by mononuclear cells, of which a fair number are eosinophils.

The pathologic diagnosis was eosinophilic granuloma of the stomach with periarteritis.

After this diagnosis was made a blood count revealed 5 per cent eosinophils. The other findings were quite normal. Serologic examination for syphilis was negative. There was no clinical evidence of any allergic manifestations. The patient, after a stormy convalescence, made an excellent recovery.

REFERENCES

- 1 Morgagni J. B. *Recherches anatomiques sur le siege et les causes des maladies traduites du latin par MM. A. Desourmaux et J. P. Destouet*, Paris, 1820-1824.

- 2 Cruveilhier, J · Anatomie pathologique du corps humain, ou description avec figures lithographiées et coloriées, des diverses altérations morbides dont le corps humain est susceptible, vol 2, chap 30, Paris, Baillière, 1835-1842
3. Rokitansky, C A Manual of Pathologic Anatomy, English translation, vol 2, 1885
- 4 Brinton, W The Diseases of the Stomach, with Introduction on Its Anatomy and Physiology, being lectures delivered at St Thomas' Hospital, London, Churchill, 1859
- 5 Dieulafoy, G Clinique médicale de l'Hôtel Dieu de Paris, vol 3, pp 219-237, Paris, Masson, 1898-1899
- 6 Velde, G · Die Magenschleimhaut bei Achylia gastrica und perniziöser Anämie Ihr Verhalten auf vegetative Reize, Ergebn med Strahlenforsch 6 347, 1933
- 7 Hansen, K, and Simonsen, M Röntgenologische Beobachtung und Darstellung der allergischen Gastritis und des allergischen Pylorospasmus, Röntgenpraxis 9 145, 1937
- 8 Scherer, H J Über Riesenfaltenbildung der Magenschleimhaut, Frankfurt Ztschr Path 40 357, 1930
- 9 Spriggs, Edmund I, and Marxer, O A Polyps of the stomach and polypoid gastritis, Quart J Med 12 1, 1943
- 10 Cole, L G Hypertrophic gastritis, M Clin North America 17 1, 1933
- 11 Freedman, E, Glenn, P M, and Lap-
ply, T C Chronic gastritis simulating gastric carcinoma, Arch Int Med 71 23, 1943
- 12 Hinkel, C L Hypertrophic gastritis simulating intramural tumor of the stomach, Am J Roentgenol 53 20, 1945
- 13 Maimon, S N, Bartlett, J P, Humphreys, E M, and Palmer, W L Giant hypertrophic gastritis, Gastroenterology 8 397, 1947
- 14 Berne, C J, and Gibson, W R Giant hypertrophic gastritis, West J Surg 57 388, 1949
- 15 Bartlett, J P, and Adams, W E · Generalized giant hypertrophic gastritis simulating neoplasms, Arch Surg 60 543, 1950
- 16 Grime, R T, and Whitehead, R Giant hypertrophic gastritis simulating malignant disease, Brit J Surg 39 244, 1951
- 17 Lyall, D, and Leider, H J Prepyloric gastritis simulating gastric carcinoma, New York State J Med 50 1483, 1950
- 18 Weems, H Stephen Emphysematous gastritis, Am J Roentgenol 55 588, 1946
- 19 Kaijser, R Zur Kenntnis der allergischen Affektionen des Verdauungskanal vom Standpunkt des Chirurgen aus, Arch klin Chir 188 36, 1937
- 20 Barrie, H J, and Anderson, J C Hypertrophy of the pylorus in an adult with massive eosinophil infiltration and giant cell reaction, Lancet 2 1007, 1948
- 21 Sikl, H Eosinophile Myokarditis als idiosynkrasischallergische Erkrankung, Frankfurt Ztschr Path 49 283, 1936
- 22 Vanek, J Gastric submucosal granuloma with eosinophilic infiltration, Am J Path 25 397, 1949
- 23 Spencer, J R, Comfort, M W, and Dahlin, D C Eosinophilic infiltration of the stomach and bowel associated with pyloric obstruction and recurrent eosinophilia, Gastroenterology 15 505, 1950
- 24 Domach, I, and McKeown, K C A case of eosinophilic gastritis, Brit J Surg 39 247, 1951
- 25 Ruzic, J R, Dorsey, J M, and Huber, H L Gastric lesion of Loeffler's syndrome, J A M A 149 534, 1952

Hypertrophy of the Pylorus

ETIOLOGY

The subject of hypertrophic stenosis of the pylorus is of considerable clinical and roentgenologic importance. There are a number of causes which may underlie the development of a condition of this nature, and a survey of these factors will aid considerably in the interpretation of the roentgenologic findings in such cases.

1 Hypertrophic pyloric stenosis may be of congenital origin. One of the earliest descriptions of pyloric occlusion coming under this classification was given by Calder.¹ In addition to the fact that this condition is present at birth, however, it is important also to realize that, although of congenital origin, such stenosis may last well into adult life. Thus Maier² described the autopsy findings in 31 such cases; the ages of the patients varying from 12 to 80 years. There was a history of epigastric pain and vomiting going back to early infancy. The stomach was dilated, the antrum funnel shaped and narrowed, and a constriction 2 to 8 cm in length represented the pyloric canal. The pyloric sphincter was hypertrophic and in some cases the rest of the stomach as well. Heaped up folds in the pyloric canal were an additional factor in exaggerating the obstruction. There was no evidence of ulceration or inflammation.

2 Another cause of hypertrophic pyloric stenosis is an underlying gastritis. In such cases the narrowing is due to an actual inflammatory hyperplasia, involving the mucosa and submucosa associated with hypertrophy of the muscular layer, the entire

pathologic process leading to various degrees of pyloric occlusion.

3 The presence of an ulcer, particularly at the pylorus, may lead to stenosis as a result of an associated chronic gastritis exaggerated by spasm ultimately leading to thickening of the pyloric muscle. This tendency for the pyloric muscle to become hypertrophied secondary to ulcer was shown by the anatomic investigation of Horwitz, Alvarez and Ascanio.³ They found the pyloric muscle in 47 individuals without organic disease of the stomach or duodenum to vary from 3.8 to 8.5 mm in thickness. It generally became hypertrophied in the presence of gastric ulcer. In some cases the ulcer as the initiating factor may disappear, and the tendency to pyloric hypertrophy may then proceed of its own momentum.

4 The possibility must also be considered that spasm of extragastric origin may be a factor in the development of pyloric hypertrophy. Thus Thomas and Wheelon⁴ showed the reflex origin of pylorospasm secondary to stimulation of afferent nerves of various viscera. Pylorospasm of extragastric origin has long been recognized as arising on the basis of such lesions as gallbladder disease. The role of the autonomic system in the production of antral spasm was referred to previously in the description of the experimental work of Klee. Such spasm of long standing may well be a factor in the eventual development of pyloric hypertrophy. The original cause may subside, but the pyloric hypertrophy may remain.

ROENTGEN DIAGNOSIS

In the congenital variety in the newborn, roentgen examination will disclose marked enlargement of the stomach and particularly abnormal delay in emptying. An almost complete retention of barium may be present at 6 hours, and frequently also at 24 and 48 hours and even longer. The pyloric canal, when visualized, will appear markedly elongated. Meuwissen and Sloof⁵ showed that although in normal children the pyloric canal in its longest dimension averages only from 1 to 2 mm, in infants with congenital stenosis the pyloric canal may show elongation by roentgen examination beyond the limits of 6 or 7 mm.

While the diagnosis of hypertrophic pyloric stenosis in infancy is primarily clinical, roentgenographic examination may perform a useful function. In the application of the roentgen method the following points are important:

- 1 The stomach should be as empty as possible before the barium suspension is introduced, otherwise small amounts of retained food or mucus may occlude the pyloric opening and prevent the escape of the ingested barium through the pathologically narrowed segment. The occlusion of the lumen produced by the hypertrophied pyloric muscle, particularly when exaggerated by an associated gastritis, may be such as to preclude the possibility of the roentgenographic delineation of the constricted segment. Therefore, it is readily understandable that even a very small amount of nonfluid material may increase the difficulties to a degree which would completely prevent escape of the barium even during a prolonged period of observation. Therefore, prior to examination, the stomach should be emptied as thoroughly as possible by means of suction through a nasal catheter. I think that it is also wise to examine the child in the morning after a period of 12 hours or more during which no food has been administered.

- 2 The barium should be administered as

a watery suspension. It may be sweetened slightly with sugar, if that will encourage the infant to drink it. The barium should not be mixed with any milk formula since the food may cause mechanical occlusion of the lumen. The suspension should be administered by mouth whenever possible, the swallowing being watched carefully under fluoroscopic guidance. Only if the child rejects the mixture and it cannot be made to take it by mouth should it be introduced into the stomach through a nasal catheter. The reason is that fluoroscopic visualization of the structure and the functional behavior of the esophagus may yield valuable information as to the cause of persistent vomiting in some obscure cases. Not only may the cause of the vomiting be due to a primary abnormality of the esophagus but it may be coexistent with an associated hypertrophy of the pylorus. The amount of the barium suspension to be administered in order to fill the stomach in any individual case may also be determined best by fluoroscopic observation.

- 3 Therefore, fluoroscopy must not be considered as merely an incidental part of the examination. In addition to the opportunity it affords for a study of the esophagus, the fluoroscopic visualization of the stomach may be extremely helpful for a number of reasons:

- A It enables us actually to see the vigorous peristalsis which is a characteristic of the disease.

- B The child may be placed in the ideal position which will show best the pyloric segment and the roentgenogram taken with spot film technic.

- C Also, the auspicious moment for making the exposure may be noted, that is during a phase of markedly hyperactive peristalsis when it may be assumed that barium has been forced through the narrowed pyloric outlet. The fine line in the roentgenogram representing the area of constriction may be such as to defy detection during fluoroscopy. At times the filling of the constricted segment with barium may

be aided by gentle manual pressure on the stomach under fluoroscopic guidance. Fluoroscopies may also enable one to correlate the position of the palpable movable tumor with the distal pylorus.

4 The ideal position of the child for roentgenographic exposure is the right oblique. Sometimes in extreme right lateral position may be essential for the complete and detailed visualization of the pylorus. The prone position is more desirable than the erect. In the first place, it is easier for the child to maintain this position. Secondly, in the event that the child struggles it can be controlled more easily. Also, the barium filled stomach may empty itself more readily with the child in the prone than in the erect position.

5 With the infant in the ideal position for roentgen exploration several exposures should be made. In the first place this increases the chances of success in the demonstration of the area of constriction. Secondly, at times the length of the constricted segment may be exaggerated by an associated spasm of that portion of the stomach just proximal to the hypertrophied pylorus. Indeed at times transient spasm of the pylorus may simulate the appearance seen in actual hypertrophy. Therefore careful fluoroscopy combined with an evaluation of the appearance in the various roentgenograms will enable one to determine more accurately the length of the permanently constricted segment corresponding to the actual length of the anatomic lesion.

6 The films should be developed promptly. If the deformity of the pylorus is not shown clearly, then additional studies may be essential at intervals of 10, 20 and 30 minutes. As previously stated the obstruction of the pyloric segment produced by the tumor may be so marked as to make it impossible to demonstrate the line of communication between the stomach and the duodenum. In that event the only roentgen evidence of the lesion may be the unusual size of the stomach, evidence of exaggerated peristalsis and an abnormal de-

gree of retention at 6 hours and particularly at 24 hours.

7 In the event that the pyloric obstruction is extreme and very little barium has escaped by way of the pylorus within the first few hours, it may be advisable to empty the stomach by suction through the nasal catheter. This may avert the potential danger of a reflux of barium from the stomach into the esophagus and then by way of the hypopharynx into the lungs.

8 When the narrowed pyloric segment is actually visualized it may exhibit a number of characteristic features. Most important is the abnormal length of the segment appearing like a 'string'. The area of constriction while often quite straight, may sometimes exhibit a slight irregularity of contour. Occasionally it may appear curved in an upward direction. When the constriction is less nearly complete there may be evidence of the narrowed elongated lumen outlined by barium clinging to the sides. When the duodenal bulb is visualized an important associated feature may be the concave pressure defect to either side of its base. This is produced by the actual anatomic pressure which the tumor exerts upon the base of the bulb. The active peristaltic contraction of the stomach may aid further in the production of this deformity by the propulsion of the hypertrophied pylorus against the duodenal bulb.

A similar concave defect may be noted in the stomach to either side of the constricted segment. Here again the reason for the deformity is the pressure exerted by the proximal portion of the annular pyloric tumor. While the abnormally elongated pyloric segment is the prime diagnostic evidence, these concave deformities both proximal and distal to the tumor render further support to the diagnosis.

9 The roentgen examination may be of importance as an objective method of determining the effectiveness of medical management which may be attempted, particularly in the less severe cases. It may also be useful as a follow up procedure after

operation. Objective evidence of the degree of dilatation of the pylorus may be secured thereby and compared with studies in later life. In spite of a good clinical response, residual evidence of a pyloric deformity may be noted for many years.^{6,8} This may throw some light on the nature of the deformity of the pylorus in the adult. In some of these cases it may represent residual evidence of a mild hypertrophy of the pyloric muscle initiated during infancy.

Differentiation from the obstruction produced by duodenal atresia should offer no difficulty. In this latter condition, not only is the stomach markedly dilated, but the duodenum also shows enormous distention, with a failure of any of the barium passing distally. The pyloric region itself shows no evidence of constriction. In congenital hypertrophic pyloric stenosis, the narrowing involves the distal segment of the pylorus. Ordinarily, the duodenal bulb is not visualized because of the marked pyloric obstruction. When a small amount of barium is finally forced through into the bulb, its size will appear normal. Moreover, while the obstruction in duodenal atresia is complete, ordinarily in congenital stenosis traces of barium will be noticed to have escaped into the small intestine in the course of 24 hours.

In the adult type of pyloric hypertrophy, the appearance will vary with the nature of the underlying cause. In the absence of chronic gastritis or ulceration, the area of pyloric narrowing will be elongated and smooth in outline. The mucosal folds will appear intact, and some evidence of change in contour may be noted as the result of peristaltic activity. Because of the partial invagination of the hypertrophied pylorus into the duodenum, there may be a concavity at the base of the bulb, as described by Kirklin and Harris.⁹ Of course, such evidence of a concave appearance at the base of the bulb must be of a permanent character, since this may be observed on a single film even in normal patients. The significance of such a finding is considerably

reinforced by an elongated narrowing of the pars pylorica of a persistent nature.

In the case of hypertrophy of the pylorus described by Crymble and Walmsley,¹⁰ pathologic examination showed that the cause of the tumor at the pylorus was hypertrophy of the circular muscle limited to the pyloric canal. This was entirely separated from the normal pyloric sphincter, which did not partake in the hypertrophy. In view of this observation, Twining¹¹ believed that a radiologic aid in the recognition of this condition would be an indentation to either side of the pyloric canal marking the cleft between the pyloric sphincter and the hypertrophic prepyloric muscle. Roentgen evidence of this sharp delimitation between the pyloric sphincter and the localized area of pyloric hypertrophy is rather illusive and difficult to demonstrate with any degree of assurance.

As a result of the obstruction resulting from pyloric hypertrophy, various degrees of gastric distention and abnormal retention will result. If the obstruction is marked, an insufficient amount of barium may be forced through the pylorus for proper visualization of the duodenal bulb.

In the presence of chronic gastritis, further difficulties are introduced into the roentgen diagnosis of the pyloric hypertrophy resulting therefrom. In the first place, there is a greater tendency for the contour of the narrowed pyloric region to be irregular in outline. In addition, considerable change in the appearance of the gastric mucosa may result, due to the abnormal prominence of the mucosal folds and the presence of polypoid excrescences producing irregularly outlined translucencies in the involved segment. In such cases, particularly of an advanced character, differential diagnosis from a malignant infiltration may be difficult or impossible. The obstruction may be so marked that only a thin stream of barium may be noted leaving the pylorus, insufficient for visualizing the duodenum, and considerable gastric dilatation may ensue.

FIG 147 Hyper-
trophic pyloric sten-
osis Examination in
the extreme right
oblique position in
order to demon-
strate clearly the
abnormally long
narrowed pyloric
segment correspond-
ing to the tumor
found at operation
There was a 6 hour
gastric residue of
approximately two
thirds of the barium



When an underlying ulcer is associated with the pyloric hypertrophy, at times it may be demonstrable as a niche in the prepyloric region or on the posterior wall when the thin layer method of examination is employed. Often, however, the ulcer itself, particularly when present on the posterior wall, will go unrecognized and only the organic narrowing of the pylorus be seen. These changes will be similar to those in cases in which chronic gastritis is the underlying cause. Indeed, it is this associated gastritis rather than the ulcer itself that may be responsible for the roentgen manifestations.

Essential Roentgen Findings The essential roentgen findings in pyloric hypertrophy are therefore

- 1 Elongation of the pyloric canal
- 2 Persistence of some degree of peristaltic activity, even though this may be very superficial in character
- 3 An intact mucosal relief
- 4 In some cases a concave pressure deformity of the base of the bulb when this region is demonstrable
- 5 Secondary gastric enlargement with disturbed motor function
- 6 In borderline cases, however, as stated

above the deformity in the contour of the pylorus and the abnormal appearance of the pyloric mucosa may be of such nature as to suggest strongly the presence of malignant disease.

The markedly elongated and narrowed pylorus characteristic of congenital hypertrophic pyloric stenosis is illustrated by the following case.

R. M. male, aged 7 weeks. The mother of the baby had a normal full term easy delivery. There were two other children, one a girl 4 years old and the other a boy 2 years old, both well. There was one miscarriage. The baby weighed 6 pounds 4 ounces at birth, was breast fed and appeared to be in good health. Five days prior to admission to the hospital the child began vomiting immediately after feeding; the vomiting was projectile in character. At this time the child became constipated although previously the bowel movements had been regular and normal in appearance. Physical examination of the abdomen showed visible peristaltic waves from left to right; occasionally they seemed to cause pain. There were no palpable masses or other abnormal findings. Because of the clinical impression of pyloric stenosis as well as the roentgenographic evidence obtained on examination of the upper digestive tract a diagnosis of hypertrophic pyloric stenosis was made. As part of a conservative medical regi-



FIG 148 Markedly elongated and constricted pylorus in an operatively confirmed case of hypertrophic pyloric stenosis

men, the child was treated with Eumydrin but without any particular result. Therefore, a Ramstedt operation was done. A large olive-sized pylorus was palpable.

Roentgenographic examination (Fig 147 in the extreme right oblique position) illustrates a number of the significant features in the examination of the stomach when hypertrophic pyloric stenosis is suspected. Note that the infant was in the extreme right oblique position in order to obtain optimum visualization of the pyloroduodenal segment. The barium was introduced into the stomach through a nasal catheter. Note the abnormally long narrowed pylorus corresponding to the olive-sized tumor found at operation. In addition, there was a retention of approximately two thirds of the barium in the stomach at 6 hours.

An additional example of a markedly elongated narrowed pylorus in an operatively confirmed case of hypertrophic pyloric stenosis may be noted in Figure 148. Note the upward swing of the threadlike elongated pylorus.

While the demonstration of the abnormally elongated pylorus is obviously the ideal procedure, it cannot always be accomplished. The opening of the pylorus may be so minimal as to defy homogeneous filling of the threadlike canal during prolonged periods of observation. In that event, the clinical diagnosis will be substantiated only

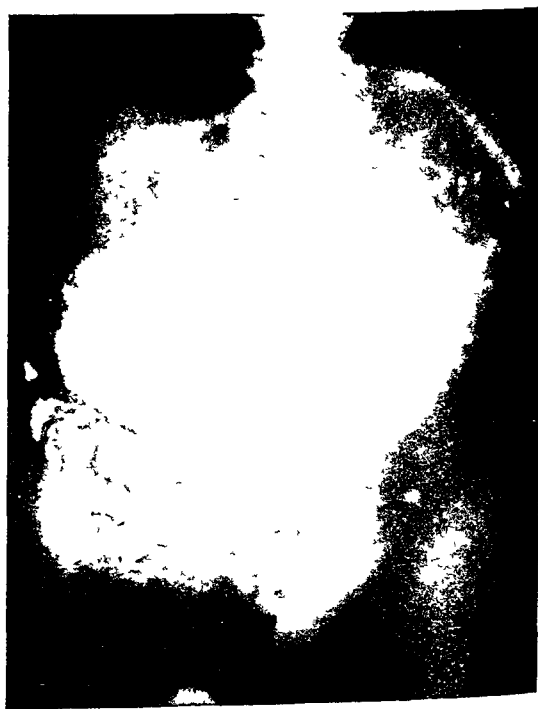


FIG 149 (A, *Left*) Congenital pyloric stenosis. Appearance after filling the stomach with barium. (B, *Right*) Same patient. Appearance 6 hours later. Note the marked gastric retention.

by the indirect evidence resulting from the obstruction, namely, the marked enlargement of the stomach and the abnormal delay in emptying. These features are illustrated by the following cases:

R. C., a 6 week old colored male child, had been well and gaining weight until he was just under 3 weeks of age, when he began to vomit his feedings. The vomiting occurred about a half hour after meals at first but at the time of hospitalization it occurred from 10 to 20 minutes after feedings. It was projectile in character. No blood was ever seen in it and there was never any blood in the stool.

On physical examination, the outline of the stomach could not be made out, nor was there any visible peristalsis. A small mass about 1 by 2 cm. was palpable in the right upper quadrant.

At operation, there was a firm tumor, 2 cm. in diameter, at the pylorus. An incision 1 inch long was made through the circular muscle of the tumor down to the mucosa. The muscle was spread with forceps and the mucosa was allowed to bulge into the opening.

The pathologic diagnosis was congenital hypertrophic pyloric stenosis.

The roentgen examination showed that the stomach was of increased size, but there was no deformity of contour. The pyloric ring and

duodenal bulb could not be visualized. No barium was noted leaving the stomach throughout the examination (Fig. 149 A). At the end of 6 hours (Fig. 149 B), the stomach appeared even larger than at the preceding examination, the pyloric portion extending over to the extreme right. Only a comparatively small amount of barium escaped from the stomach and was present in the small intestine.

The roentgen diagnosis was congenital hypertrophic pyloric stenosis.

The stomach may become comparatively huge as the result of a congenital hypertrophic pyloric stenosis.

G. E., female, aged 10 weeks. This colored baby girl began vomiting at the age of 6 weeks. From then on she vomited practically after every feeding. Marked peristalsis was noted, passing from left to right in the upper abdomen. A small, hard, olive shaped area was palpated in the right upper quadrant. At operation the pyloric ring was found to be hard and cartilagenous (hypertrophic pyloric stenosis). A Ramstedt operation was performed.

The roentgen examination (Fig. 150) showed a hugely dilated stomach with most of the barium present distally and the rest of the stomach outlined mainly by air. None of the barium had escaped into the small intestine.



FIG. 150 Hypertrophic pyloric stenosis. Note the marked gastric enlargement. Most of the barium has been forced into the distal portion of the stomach. Note also the unusually prominent greater curvature of the stomach, probably due to hypertrophy of the wall.

tive The physical examination was essentially negative

At operation, a firm, even, stony-hard, infiltrating mass along the lesser curvature of the stomach was found, posteriorly from the midportion of the stomach to and apparently involving the pyloric region A few small, firm nodes were present in the prepyloric region posteriorly The stomach was only moderately

dilated The liver was smooth and glistening A partial gastrectomy was done

Macroscopic examination of the specimen revealed the pylorus to be practically occluded because of its greatly thickened walls Immediately next to the pylorus in the greater curvature was a well-defined, yellowish-white, dense, depressed, smooth, diamond-shaped area resembling an ulcer 1.5 by 1 cm in size

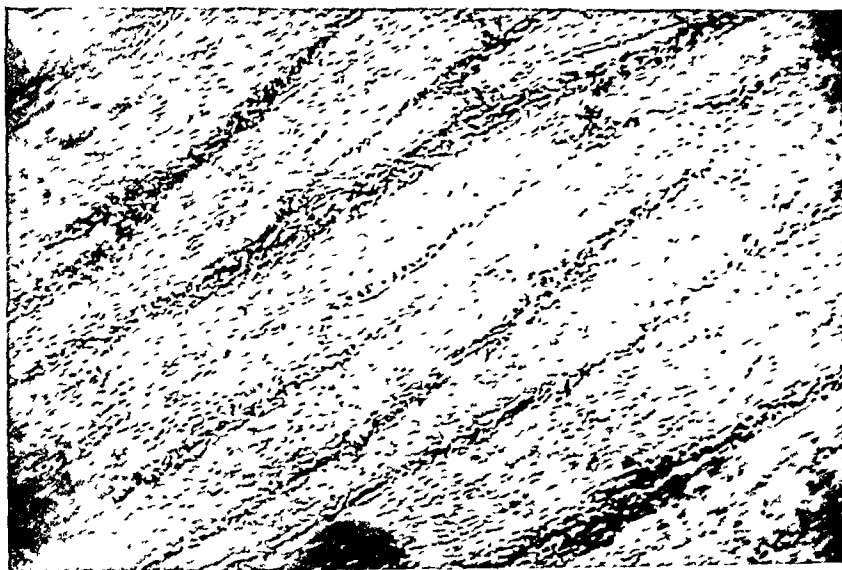


FIG 152 (C, *Top*) Same patient as in Figure 152 A and B, showing the presence of interstitial gastritis (D, *Bottom*) Same patient, showing marked tubular narrowing at the pylorus



(Fig 152 A and B) Adjacent to this area the mucosa was reddish brown. A section through the ulcer showed the overlying tissue to be dense, white and homogeneous.

The report of the microscopic examination (Fig 152 C) was as follows: Section through the ulcer shows the base of the ulcer to be covered with necrotic fibrin and cellular elements beneath which is seen a rather marked vascular fibroblastic reaction which extends down to the muscularis. This area is also infiltrated with many polymorphous plasma cells and eosinophils. The edge of the ulcer shows atrophic mucosa in which a similar cellular reaction is present. Section through the pylorus of the stomach shows atrophic glandular elements with marked interstitial fibroblastic proliferation, vascularization and cellular infiltration similar to that described above. The subjacent tissue consists of moderately vascular, dense fibrous tissue. The muscularis in this region shows a marked round cell infiltration between the bundles. Section taken through the serosa and including the muscularis along the lesser curvature shows a similar marked cellular reaction to be present.

The diagnosis was chronic interstitial gastritis with superficial simple ulceration of the stomach; there was no histologic evidence of malignancy.

The roentgen examination revealed a marked annular narrowing of the pyloric portion of the stomach of fairly smooth contour.

There was marked obstruction to the flow of barium beyond this region. The stomach was of increased size. The conclusion was 'pyloric obstruction of organic origin due to an infiltrating tumor' (Fig 152 D).

The pathologic examination mentioned above showed that the pyloric narrowing was due to a benign hypertrophic process associated with a pyloric ulcer. Roentgenologically it would be impossible to determine the exact nature of the pathologic process, since the appearance in the roentgenogram might well have been produced by a malignant infiltration.

The next case of pyloric stenosis shows many features of interest.

W. B., aged 52. This patient had been complaining of recurring attacks of pain, localized to the epigastrium, coming on 1 hour before mealtime and relieved by the ingestion of food. There was no belching, vomiting or sour eructation. There had been only slight loss of weight during the 11 months of observation prior to surgical intervention. He was put on the Sippy regimen and made an excellent response only to have his symptoms recur within 9 months. The physical examination was essentially negative.

At operation a partial gastrectomy was



FIG 153 A Appearance of the resected specimen showing an ulcer in a case of marked pyloric narrowing

performed On inspecting the specimen, an ulcerated area of the pylorus was found by the surgeon this he felt might be either an ulcer or an early carcinoma, but more likely the former This was confirmed by pathologic examination Because of the roentgen diagnosis, I am quoting the pathologic report in full

The report of the macroscopic examination was "Specimen is a portion of stomach It

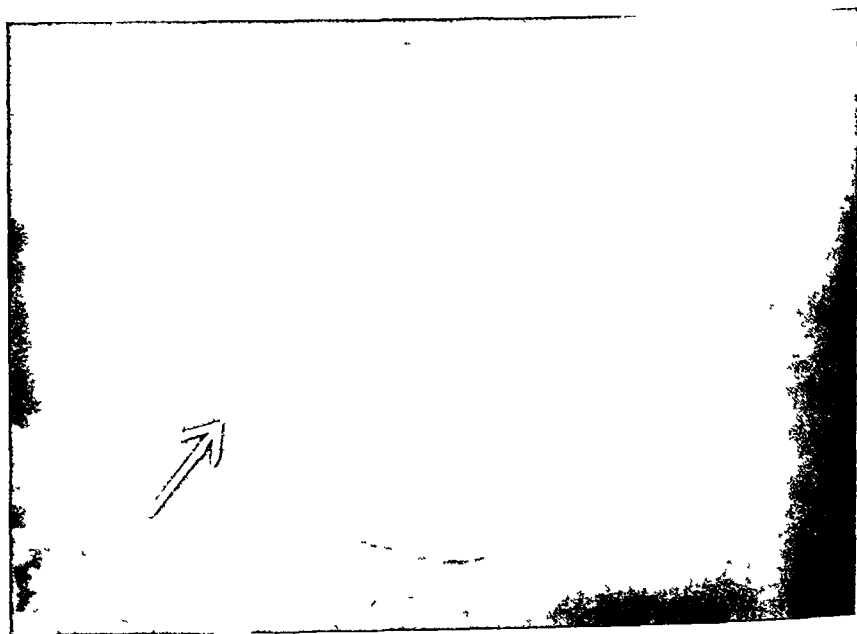
measures 65 mm in length and varies in width from 35 to 60 mm On opening this portion of the stomach, the interior appears negative except for one oval area measuring 22 mm in length and about 8 mm in width This is an ulcerated area about 5 mm in depth and showing a smooth base The remaining mucosa appears negative The wall of the stomach corresponding to this ulcerated area is slightly thickened and shows a small amount of fibrosis (Fig 153 A) "

The microscopic report was as follows "Sections of the tissue reveal a portion of stomach wall showing an area of ulceration where the mucosa, submucous layer and a portion of the muscular coat are absent The surface of the ulcerated area is covered by a large amount of necrotic granular material The tissue around the ulcerated portion shows extensive fibroblastic proliferation, with numerous foci of lymph follicles, lymphocytes, plasma cells and eosinophiles Diagnosis peptic ulcer (Note there is no evidence of malignancy in this tissue) " (Fig 153 B)

Roentgenologically, a diagnosis of carcinoma of the pylorus had been made, based on the marked irregular narrowing of the antrum persistent throughout prolonged fluoroscopic examination and on all films In addition, several translucent areas were noted in this region, which suggested destruction of the mucosa due to new growth



FIG 153 (B, Top) Same patient as is shown in Figure 153 A Microscopic appearance (C, Bottom) Same patient Pyloric stenosis



The roentgen appearance was evidently the result of the extensive fibroblastic infiltration that was reported by the pathologist (Fig 153 C).

Hypertrophic stenosis of the pylorus may be present even though the ulcer is some distance away as noted in the next case.

G. S. male, aged 53. The patient gave a 1 year history of intermittent attacks of epigastric pain coming on 1½ hour after meals lasting from 4 to 5 hours and associated with occasional vomiting. There was no blood in the vomitus or stool. There was only slight weight loss. The pain persisted in spite of medical treatment. Physical examination of the abdomen was essentially negative.

The operative findings revealed an ulcer on

the lesser curvature about 5 cm proximal to the pylorus. The gastrophrenic omentum in this area was thickened and edematous. There was a marked thickening around the pylorus but no definite ulcer. This thickening had caused considerable pyloric stenosis.

A partial gastrectomy was done and the distal two thirds of the stomach removed.

The pathologic report was gastric ulcer, no malignancy, pyloric hypertrophy.

Roentgen examination (Fig 154) revealed a niche in the lesser curvature at the junction of the pars pylorica and the pars media with the roentgen characteristics of a benign ulcer. In addition there was a tubular narrowing of the prepyloric segment of essentially smooth contour without any very sharp demarcation from the rest of the stomach, interpreted as due to associated pyloric hypertrophy.

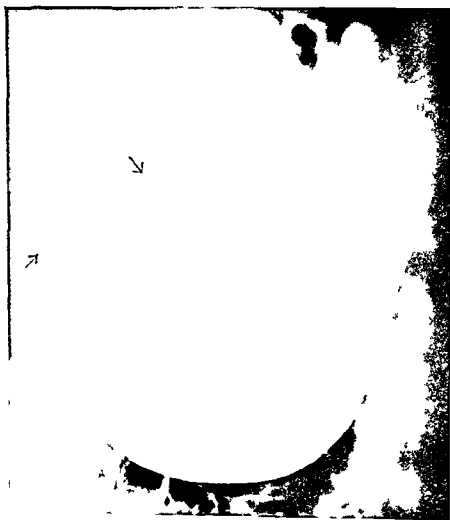


FIG 154 Hypertrophy of the pylorus associated with gastric ulcer

REFERENCES

- 1 Calder, J Two examples of children born with preternatural conformations of the guts, *Med Essays and Observations* 1 203, 1733
- 2 Maier, R · Beitrage zur angeborenen Pylorusstenose, *Vuchow's Arch path Anat* 102 413, 1885
- 3 Horwitz, A, Alvarez, W C, and Ascanio, H · The normal thickness of the pyloric muscle and the influence on it of ulcer, gastroenterostomy and carcinoma, *Ann Surg* 89 521, 1929
- 4 Thomas, J E, and Wheelon, H The nervous control of the pyloric sphincter, *J Lab & Clin Med* 7 375, 1922
- 5 Meuwissen, T, and Sloof, J Roentgen examination of pyloric canal of infants with congenital hypertrophic pyloric stenosis, *Am J Dis Child* 48 1304, 1934
- 6 Runstrom, G On the roentgen-anatomical appearance of congenital pyloric stenosis during and after the manifest stage of the disease, *Acta Paediat* 26 383, 1939
- 7 Andresen, K Roentgenologic follow-up examination in congenital pyloric stenosis after the manifest stage, *Acta paediat* 27 334, 1940
- 8 Olnick, H M, and Weems, H S Roentgen manifestations in infantile hypertrophic pyloric stenosis, *J Pediat* 34 720, 1949
- 9 Kirklin, B R, and Harris, M T Hypertrophy of the pyloric muscle of adults a distinctive roentgenologic sign, *Am J Roentgenol* 29 437, 1933
- 10 Crymble, P T, and Walmsley, T Hypertrophy of the pylorus in an adult, *Brit J Surg* 20 602, 1933
- 11 Twining, E W Chronic hypertrophic stenosis of the pylorus in adults, *Brit J Radiol* 6 644, 1933

Gastric Ulcer

Although descriptions of gastric ulcer checked at autopsy can be found in the literature from the time of Marcellus Donatus in 1586, it was not until the classic descriptions of Morgagni¹ Abercrombie² and particularly Cruveilhier³ in 1829 that simple chronic ulcer of the stomach became clearly recognized as a definite pathologic entity.

Cruveilhier's first patient was a coal carrier 23 years old, who was brought to the hospital in a moribund state on December 15, 1829. The preceding morning while carrying a bag of coal he had experienced a sudden attack of excruciating abdominal pain. He died 3 hours after his admission to the hospital. The autopsy revealed the cause of his death to have been the perforation of a round ulcer of the stomach at the pyloric orifice. Cruveilhier also included other examples of characteristic simple chronic ulcers of the stomach. One of his pictures was of an unusually large gastric ulcer which had perforated through all the coats of the stomach, exhibiting a considerable portion of the pancreas at the floor of the ulcer.

Cruveilhier's description of chronic simple ulcer of the stomach was epoch making. Such an ulcer according to him consists of a loss of substance, usually circular and of punched out appearance with a grayish base. It is of varying size, is almost always solitary, and is usually situated either at the lesser curvature or on the posterior wall of the stomach. It burrows through the walls of the stomach and if this process is not walled off by the formation of adhesions the ulcer goes on to perforation with the

escape of gastric contents into the peritoneal cavity. In his opinion the primary lesion was an erosion of the mucosa due to the ulcerating inflammation described by Hunter. Finally, this erosion becomes a true ulcer with all its pathologic characteristics. Cruveilhier also stated that simple ulcer of the stomach shows none of the attributes of cancer—a fact that is best demonstrated by its curability and tendency to heal under simple therapy. Essentially, his description of the pathology of gastric ulcer holds to this very day.

LOCALIZATION AND FREQUENCY

A factor which may explain the predilection of ulcer for the lesser curvature of the stomach may be the nature of the circulation in this region. The blood vessels are smaller here and make fewer anastomoses. Similarly, in the bulb it is stated the arteries are few in number and small in size and do not anastomose as freely as elsewhere in the duodenum. Such peculiarities may possibly make them more susceptible to thrombosis from infective emboli and may lead to the more frequent occurrence of chronic ulcer. The acute ulcer may occur elsewhere throughout the stomach and duodenum, but it heals more readily.

In addition to peculiarities of the blood supply, the distribution of the lymphoid follicles may also play a part in the greater susceptibility of certain regions of the stomach and the duodenum to formation of ulcers. Such follicles are much more common in the pyloric portion of the stomach than in the cardiac region. They also are encountered more frequently on the lesser

curvature than on the greater curvature and also predominate in the duodenal bulb. An abscess developing in such a follicle may perhaps be a factor in the eventual origin of an ulcer, as originally suggested by Cruveilhier.

Aschoff⁴ evoked a mechanical factor in his endeavor to explain the localization of ulcer in the region of the lesser curvature. He described two types of mucosal erosions: those in the fundus and those along the gastric pathway, or "Engpasse" of Forssell. Whereas the fundal erosions healed rapidly, chronic ulcers, practically speaking, developed only from the second type of erosions. In experiments on rabbits by Dr Yano which Aschoff recorded, losses of substance produced by burns on the gastric pathway healed with much more difficulty or not at all, while, in the fundus, the artificially produced erosions healed fairly rapidly. As a result, although only a faint scar remained to mark the site of a fundal erosion, an actual chronic ulcer might develop in the gastric pathway. Aschoff believed that an erosion of the "Engpasse" is in more direct contact with the acid gastric juice and is subjected to greater mechanical injury by the peristaltic motion in that region, and also that the protective mucus which is secreted in the fundus and is an aid in healing is generally not present in the gastric pathway.

On the basis of such conceptions, Aschoff found it necessary to invoke a mechanical factor in the structural and functional peculiarities of the "Engpasse" to explain the development of chronic ulcer from a mucosal erosion. A survey of these various factors gives us a clearer insight into the reasons why, roentgenologically, peptic ulcer is so commonly found at or near the lesser curvature of the stomach.

Sturtevant and Shapiro⁵ made a careful analysis of 7,700 autopsy records at Bellevue Hospital during the period from 1904 to 1922. They found 120 gastric ulcers, of which 34 were healed. In this same series were 44 duodenal ulcers, of which 9 were

healed. Of the 86 cases of open gastric ulcer, there were 4 in which an open duodenal ulcer was also present. Since these were also included in the series of open duodenal ulcers, there was thus a total of 117 cases of unhealed gastric or duodenal ulcer or both. Two per cent of the total of autopsy cases in the Bellevue Hospital series showed the presence of a gastric or a duodenal ulcer, either active or healed. Gastric ulcers represented 1.5 per cent of the total.

Fenwick, on the basis of 47,912 autopsies from various sources, mainly continental, found 4.2 per cent of gastric ulcers. William H. Welch arrived at an estimate of 5 per cent based mainly on continental reports, although in 800 autopsies at Bellevue Hospital he found only 6 gastric ulcers. Lockwood also found 6 gastric ulcers in 1,000 autopsies at Bellevue Hospital, and Harlow Brooks reported 9 cases in another series of 1,000 at the same institution.

Sturtevant and Shapiro found combined gastric and duodenal ulcer five times in a series of 159 cases of ulcer. This represented an incidence of about 3 per cent. Of the duodenal ulcers, 11 per cent had a coexisting gastric ulcer. Of the 120 gastric ulcers, 86 were single, the rest were multiple, there being 12 in one patient. They quoted Berthold, who described a patient who had 34 ulcers, and the patient cited by Lange, in whom the ulcers were so numerous they could not be counted. Of the 120 gastric ulcers, 21 were active. Five acute ulcers were found in infants. In 49 cases of simple gastric ulcer the size of the ulcer varied from less than 0.5 cm. to 5 cm. by 6 cm. The authors found that as ulcers increase in number, they are apt to decrease in size.

Of the total number of gastric ulcers, 9 were on the anterior surface, 27 on the posterior surface, 41 on the lesser curvature, and 19 on the greater curvature. Eight were at or near the cardia, and 15 others were within 8 cm. of the cardia. Of the total, about 76 per cent were near the pylorus, 12 per cent near the cardia, and 12 per cent in the midgastric zone.

With regard to the factor of sex, 89 of the patients with gastric ulcer were males and 51 females. This is approximately a ratio of 3 to 1. Thirty-four of the patients with duodenal ulcer were males, 10 were females. This again is a ratio of about 3 to 1.

The greater frequency of gastric ulcer as compared with duodenal ulcer as demonstrated in our Bellevue Hospital autopsy records was substantiated also in the autopsy studies by Hart⁶ and by Portis and Jaffe.⁷

Such findings are contrary to surgical as well as roentgenologic findings. Even an average experience with the roentgenology of peptic ulcer shows that duodenal ulcer preponderates considerably in frequency as compared with roentgen evidence of gastric ulcer. The reason why duodenal ulcer appears to be much more common than gastric ulcer roentgenologically and surgically, can perhaps be found in the greater tendency of acute gastric ulcers to heal spontaneously. Also some of these gastric lesions may be so superficial as to defy detection roentgenologically.

ROENTGEN DIAGNOSIS

A revolutionary advance in the recognition of the presence of gastric ulcer resulted from the development of the roentgen method of examination. In 1906, Hemmeter⁸ published the results of his researches. He produced ulcers in rabbits and cats experimentally by creating a defect in the gastric lining. He removed a ring of tissue, 1 cm in diameter, down to the circular layer of muscle; he then coated this denuded surface with bismuth subnitrate and, after closing the abdomen, was able to visualize the region by means of the roentgen rays.

Hemmeter then endeavored to demonstrate in a similar manner the presence of such experimentally produced denudations by introducing a bismuth suspension to spread over the lesion. This could be done provided that less than 48 hours had elapsed for he found that the base of the ulcerated area was rapidly covered with

granulation tissue. In a like manner he endeavored to visualize ulcers in human subjects by having the patient drink a suspension of bismuth in water on an empty stomach. Owing to the fact that the chronic ulcer forms a crater, frequently covered by an agglutinating transudate, he believed that this would mechanically cause the bismuth to adhere and become demonstrable with the roentgen rays. He succeeded in this in three cases. One of the patients in whom an ulcer was diagnosed in this manner was operated on by Dr. John B. Deaver, and the roentgenologic findings were confirmed.

Reiche⁹ in 1909 had occasion to examine, by means of roentgen rays, a patient, aged 47, who had a history of serious gastric disturbance of several years' duration. He noted in the middle of the lesser curvature of the bismuth-filled stomach a small, mushroom-shaped protuberance. After 7½ hours the stomach was still almost entirely filled, but it emptied at 20 hours. At this time the projecting area was also free of bismuth. The patient vomited coffee-ground material and passed tarry stools. At autopsy, an ulcer of the lesser curvature was found; this was round, about the size of a quarter, deeply penetrating, and had smooth margins. The floor of the ulcer was formed by the pancreas, and within it was a vascular lumen closed by a fresh thrombus. The depth of the ulcer in the extirpated organ was not as great as one might have been led to believe from the size of the pouch in the roentgen picture.

In this remarkable paper Reiche proved the following points of a fundamental character:

- 1 That a practical method of diagnosing chronic gastric ulcer is by demonstrating a pouchlike projection of the lesser curvature of the stomach seen in the roentgenogram. This was verified with absolute certainty by autopsy.

- 2 That although the ulcer was not present at the pylorus and no constriction was noted in that region, a serious dis-

turbance in gastric motility resulted, giving rise to retention to a pathologic degree

3 That the actual size of the ulcer did not correspond to the apparent size as seen in the roentgenogram This Reiche explained by assuming that the increased intragastric pressure pushed out the nonresistant ulcer-bearing area, devoid of its muscular layers and thereby excluded from peristaltic activity

Haudek¹⁰ in 1910 elaborated upon and popularized the roentgen method in the diagnosis of ulcer He verified its practical value in numerous cases that were proved to be correct by surgery or autopsy He summarized the roentgenologic evidence as follows (1) a diverticularlike projection of the stomach, generally of the lesser curvature, (2) a gas bubble above it, when the patient remained erect, (3) a residue of bismuth in it, visible for a protracted period, (4) the inability to influence this area by palpation Of all these, however, the first (*the presence of a niche*) is the only absolute diagnostic criterion

The Niche While the term "niche" historically refers to the roentgen appearance of the perforating type of ulcer, it is at present used more universally to apply to the pocket produced by ulcers regardless of the degree of penetration or pathologic destruction of the layers of the stomach

The most important evidence of ulcer is obviously the niche, which is the roentgen counterpart of the ulcer itself Occasionally, indirect signs may be of value as evidence of a suggestive character The larger the number of indirect signs the greater is, of course, the probability of a correct diagnosis However, a diagnosis based on such evidence cannot be made with absolute certainty since these findings may be mimicked by disorders of a functional nature It is for this reason that every effort should be made to demonstrate the niche itself, which is the ultimate diagnostic criterion of ulcer

In examining the stomach for roentgen evidence of ulcer, it is important to remember that while such lesions are usually at

or near the lesser curvature, they may also be on the posterior wall some distance away and almost anywhere in the stomach, from the cardia to the pylorus

The important point regarding this fact is that the stomach must be examined through every angle of obliquity in order to determine whether the niche of an ulcer is actually present A patient may be examined with the rays directed postero-anteriorly, that is to say, with the patient facing the fluoroscopic screen, and no deformity of the lesser or the greater curvatures of the stomach may be noted in this position However, if this same patient is turned gradually so that his right side is placed against the screen (first oblique position), eventually the niche may appear as a projection of the lesser curvature border Sometimes the patient may have to be turned until his position is practically lateral to the screen before any such evidence of niche formation is brought clearly into view This means, of course, that the ulcer is present on the posterior wall of the stomach The patient should also be turned in the opposite direction so that the left side is against the screen (second oblique position) In some cases, an ulcer on the posterior wall may then be noted as a projection of the greater curvature Moreover, the change from the vertical to the prone position, or vice versa, may make a difference in the ability to visualize the niche

In addition to examination of the stomach through every degree of obliquity, mucosal relief studies by the thin-layer method may also be essential for the recognition of a lesion on the posterior wall so centrally located that it cannot be made to project from either the lesser or greater curvature borders of the stomach The mucosal technique in such cases may show the presence of an ulcer otherwise completely obscured by the large amount of barium anterior to it It is for this reason that, in examining a stomach for a suspected lesion, under fluoroscopic control, the patient should be instructed to swallow only one mouthful

of the barium suspension at a time. After this is spread manually over the mucous membrane, the lesion may be brought into focus. In those cases in which special mucosal studies are desired, the technic is recommended which is outlined in the chapter on the study of the normal gastric mucosa.

As a rule, the niche is rounded and has a comparatively smooth outline. In rare cases the contour may be irregular, even though the lesion is benign, as the result of the protrusion within it of a blood vessel, or shortly after a subacute perforation when a thin stream of barium may be noted extending somewhat beyond the confines of the niche itself. Barium within the niche may show an irregular distribution due to the character of the surface of the lesion resulting from various gradations of destruction and regeneration of the tissues within the ulcer itself. Intrusion of pancreatic tissue within an ulcer that has perforated posteriorly may be responsible for the appearance of rounded translucencies within the confines of the niche.

Of considerable practical importance is the distribution of various layers within the confines of the niche, as seen in the erect position. Thus, in some cases instead of the niche being filled with a homogeneous mass of barium, one may note the presence of three layers: (1) a dense layer of barium at the lower pole of the niche, surmounted by (2) a less dense layer with a fluid level which is (3) capped by an air bubble. As a rule a niche with an appearance of this kind signifies that the ulcer is of the perforating type—that is to say it represents an ulcer which has slowly perforated through all the coats of the stomach and is walled off by adhesions. Frequently an ulcer like this will be found adherent to the pancreas or the under surface of the liver. It is fixed in position so that palpatory manipulation of the niche behind the fluoroscope will show an absence of or marked diminution of mobility. Another important feature regarding a niche with

this three layer appearance is that, with the rarest exceptions, it indicates that the ulcer corresponding to it is benign. An exception to this rule will be found when the subject of malignant ulcer is discussed later on. At that time, also, the differential diagnosis is between a benign and a malignant ulcer will be described.

Complicating the problem of such differential diagnosis still further are a number of other features which may simulate the appearance of a niche, although ordinarily such differentiation should be a comparatively simple matter. In rare cases, a diverticulum of the duodenojejunal junction may simulate the niche of a gastric ulcer when it overlaps the lesser curvature. Careful fluoroscopic observation should make such differentiation readily possible. By turning the patient through various degrees of obliquity, the diverticulum will be found to bear no relation to the lesser curvature of the stomach itself. Careful inspection will also show that the diverticulum springs from the duodenal curve. It is only when one attempts to make a diagnosis from the roentgen appearance on a single film that such a mistake may be made.

Confusion of a niche with an atypical peristaltic wave may also occur but only when one attempts to make a diagnosis from a single roentgenogram. Obviously fluoroscopic observation, showing the inconspicuity of such a finding, and its complete obliteration by progressive peristaltic waves will eliminate any such possible diagnostic error.

At times a small amount of barium at the duodenojejunal junction as it emerges behind the posterior wall, may also simulate the niche of a gastric ulcer in the single roentgenogram. Such an area may be recognized by the characteristic appearance produced by the valvulae conniventes as well as its transient localization, particularly as determined on fluoroscopic observation, and its incorporation within the rest of the duodenojejunal curve.

A calcified area of extragastric origin in

relation to the lesser curvature of the stomach, may also be differentiated from a niche by the fact that its shadow differs from that produced by barium, it is completely separable from the stomach, and the shadow is present either prior to the administration of the barium or long after the alimentary tract has been completely emptied

Differentiation of an ulcer from a gastric diverticulum will be discussed in more detail in the chapter devoted to a description of the latter abnormality. The essential features in the differential diagnosis are

- 1 A gastric diverticulum as a rule is located on the posterior wall near the extreme cardiac end of the stomach, to the left of the entrance to the esophagus. Gastric ulcers that high in location are very rare.

- 2 A gastric diverticulum, as a rule, has a neck, extending from the stomach and communicating with the saccular dilatation. A communicating neck of this nature is not ordinarily seen with ulcer.

An important point in the roentgen demonstration of the ulcer is the possibility of determining its actual depth from the appearance of the corresponding niche. In the case of a chronic ulcer the walls of which are rigid and, in the resected specimen, feel almost cartilaginous, the size of the niche may correspond closely to the actual ulcer itself. The condition is different, however, in the case of ulcers that have not been stiffened by considerable proliferation of connective tissue. In such cases, the ulcer in the resected specimen may show considerably less depth than the corresponding niche in the roentgenogram. In order to explain this apparent discrepancy, the role of the gastric mucosa must be taken into consideration. During life, with the ulcer intact, there is a heaping up of the gastric mucosa in the wall surrounding the ulcer as a result of edema and spasm. Roentgenologically, therefore, the actual depth of the niche depends on two factors: (1) the anatomic depth of the ulcer itself, and (2) the exaggeration of this depth by the swollen

mucosa. In the resected specimen or at autopsy the increased prominence of the mucosa is not present, and the depth noted is that of the actual ulcer itself.

That discrepancy between the depth of the ulcer, as seen in the resected specimen, and the exaggerated depth, as observed roentgenologically, is dependent upon the heaping up of the gastric mucosa surrounding the site of the ulcer is brought out by the work of Konjetzny,¹¹ who treated the gastric mucosa in such a manner as to preserve the swelling of the folds and thereby demonstrated this to be the essential factor in the apparently increased depth of the niche as observed roentgenologically during life.

As for the comparative depth of the niche in the roentgenogram and of the ulcer as seen after resection, it may actually be identical, provided that proper precautions are taken. A common source of error in a comparative study of this nature lies in the fact that the resection of the ulcer may be undertaken quite some time after the original roentgen examination. During this time a certain amount of healing may have taken place, even though it is not sufficient to cause an amelioration of the symptoms. If the ulcer in the resected specimen is then compared with the original roentgenogram, considerable discrepancy in the comparative depth will be noted. For the comparison to be accurate, the roentgen examination should have been made as soon before operation as possible. Moreover, the appearance of the ulcer should be gauged immediately after resection. Otherwise, as the specimen gets older, it may undergo changes which cannot then be accepted as indicating the true depth of the lesion during life.

A question often asked is whether one can determine from the roentgen appearance of the niche itself the extent to which an ulcer has involved the various coats of the stomach. Without an understanding of the role that the swollen mucosa surrounding the ulcer plays in exaggerating its depth, one might naively assume that a niche

which in the roentgenogram appears to possess considerable depth necessarily means destruction of more tissue in the wall of the stomach than has occurred in the case of a niche that is less deep. However, because of the variable nature of this extremely important factor of the heaping up of the folds of mucosa along the margin of the ulcer, no parallelism can be drawn between the depth of the niche and the degree of penetration through the layers of the gastric wall.

With regard to the most superficial ulcers, roentgen examination may prove to be entirely negative or may present only indirect evidence such as an incisura, localized tenderness and delayed gastric evacuation. Careful mucosal studies, however, may show even a superficial lesion by the demonstration of a niche. This is particularly true because the heaping up of mucosal folds at the wall of the ulcer may exaggerate the apparent depth of the lesion. Therefore, a niche that is readily recognizable roentgenologically may be caused by a lesion that is anatomically superficial in character. This is important, since it not only makes roentgen examination of a superficial ulcer possible but also explains why, at operation, the surgeon may fail to recognize such a lesion unless the stomach is opened. Since the ulcer has not penetrated deeply enough to involve the serosa, the surgeon may have considerable difficulty in locating it by external palpation alone.

Roentgenologically, the niche may appear as if completely separated from the stomach, because of the displacement of barium by the swollen mucosa surrounding the wall of the ulcer. A knowledge of the pathologic physiology underlying this phenomenon makes us realize that such apparent separation of the niche from the contour of the stomach does not necessarily mean that we are dealing with a perforating ulcer. Moreover, it enables us to understand one of the reasons why occasionally an ulcer may fail to be visualized roentgenologically. For if the heaped up folds of mucosa surrounding the ulcer have caused complete

occlusion, it may be impossible to force barium from the stomach into the ulcer itself.

This may be considered as a protective mechanism. It may possibly be a factor in diminishing the tendency toward perforation of an ulcer as a result of the increased intragastric pressure against it. Gutzeit, on the basis of gastroscopic observation, has shown that the swollen mucosa surrounding the wall of an ulcer may at times be approximated in so close a manner as to occlude the area of communication between the ulcer and the stomach. With diminution in the edema and swelling of the surrounding mucosa, the ulcer may then be recognizable both roentgenologically and gastroscopically.

Other Evidence of Ulcer. The following are among the indirect evidences of ulcer. First, there may be a localized permanent spastic incisura of the greater curvature. Though sometimes seen in association with the niche of the ulcer on the lesser curvature opposite, it may on occasion be the only evidence of abnormality. Such an incisura to be significant, should be permanent. However, the possibility of a functional origin must always be considered although in this type the incisura is more apt to be inconstant than when associated with ulcer. While such incisuras of ulcer ordinarily occupy the same plane as the ulcer itself at times they may involve the greater curvature at some distance from the lesion. The incisuras may vary in depth but as a rule are narrow and sharply defined.

Of some importance in the roentgen recognition of ulcer, in the absence of definite niche formation, is the contribution of Franke¹² who recommended the application of roentgen cinematography for the demonstration of ulcers of the stomach without niche formation. He took 16 roentgenograms in a period of 20 seconds and, by superimposing these in some cases he was able to demonstrate a small straight area on the lesser curvature which remained constant while the rest of the contour

showed alterations produced by peristaltic activity. He called this finding the *ulkus riegel* or crossbar, which he interpreted as being due to a segmental suppression of peristalsis. He applied the identical procedure to the demonstration of small areas of infiltration of the stomach which remained permanently unaltered while the rest of the contour was changed by peristaltic activity. When associated with an *incisura* of the greater curvature opposite, this local absence of peristalsis assumes even greater significance in the diagnosis of ulcer.

I have noticed such localized absence of peristaltic activity in cases in which a niche had been present originally where the ulcer apparently has gone on to healing. Possibly, scar formation and contraction of the longitudinal muscle fibers may play a role in such cases. Occasionally also, I have observed a sharp, smoothly outlined, localized region of the lesser curvature, without niche formation, at the site of an ulcer. However, with films taken through various angles of obliquity, sometimes it has been possible to demonstrate the actual niche. When the area has become stiff and superficially irregular, the possibility of malignancy must be considered.

Localized tenderness at various regions of the stomach is at best only suggestive and never by itself can be considered of great value.

Of some interest in connection with the study of the appearance of the mucosal folds in relation to ulcer is the occasional evidence which may be roentgenologically demonstrable of radiating folds at the site of the ulcer-bearing area.

The radiation is apparently the result of three factors: (1) contraction of the muscular layers at the site of the ulcer, (2) eventual scarring, in the case of the more chronic lesions, and (3) according to Forsell, swelling of the mucosal folds may be responsible as an additional factor.

Prepyloric Ulcers. While frequently ulcers in the prepyloric area can be diag-

nosed readily, their recognition is not quite so simple as in the case of ulcers at or near the lesser curvature of the *pars media*. Moreover, a differential diagnosis between the benign and the malignant nature of the lesion must be made with greater circumspection. Holmes and Hampton¹³ and Camp¹⁴ have emphasized the seriousness of this problem, although Singleton¹⁵ found no essential difficulty in the roentgen differentiation of a benign from a malignant lesion in the prepyloric area in a series of cases subjected to careful histologic confirmation.

Reasons for difficulty in the determination from a roentgen point of view of the benign or the malignant nature of a lesion in the prepyloric region are the following: (1) The detailed delineation of a small lesion in the pyloric region offers greater difficulties in this area than in the more proximal regions of the stomach. This is particularly true if the pathologic process involves primarily the posterior wall. (2) Lesions in the pyloric region are more apt to be associated with superimposed alterations of contour as a result of secondary spasm or chronic gastritis. Such processes may produce annular narrowing of the pylorus, irregularities and increased prominence of the mucosal folds, and even translucent areas within the involved segment, features differentiable with difficulty from those produced by a malignant process. This danger of misinterpretation is heightened even more if the posteriorly placed benign lesion cannot be brought clearly into the foreground of observation because of technical difficulties. (3) The problem of differential diagnosis in this region is complicated still more because of the fact that this is the area where changes produced by chronic gastritis, as such, may preponderate, where spasm of a reflex nature from an extragastric cause may develop, and where benign tumors such as polyps may occur.

For all these reasons, extreme care must be exercised in the determination of the nature of a lesion in this region. First and foremost is the value of the thin-layer

method for the detailed visualization of the mucosal structure in this area. An intact mucosa definitely favors the benign nature of the pathologic process. The niche of an ulcer, if on the posterior wall, may then more clearly be brought into view and, if it conforms in size and contour to the general features of the niche of a benign ulcer elsewhere and there is no evidence of invasion of the surrounding mucosa, such a lesion may be considered as presumably benign. In addition, alterations in the contour of the lesser and greater curvatures of the involved segment in various films would be in favor of a benign process. Re-examination and comparative studies may show sufficient alteration in the configuration of the involved area or diminution in the size of the niche, when demonstrable, to justify an attitude of conservative watchful waiting as these would definitely indicate the benign nature of the lesion.

As in the case of the niche of an ulcer elsewhere, it must be remembered that no criterion is absolutely final, and that, in rare instances, a lesion which seems to have features that would classify it as benign may eventually prove to be malignant. However, with a careful evaluation of all the details involved, it should be possible to make a differential diagnosis in the large majority of cases between the benign or malignant nature of a prepyloric lesion to act as a guide in any therapeutic approach.

Ulcer at the Pyloric Ring. The following are the important changes which may occur at the pyloric ring as the result of ulcer in this region. The pyloric ring may be angulated or slightly curved presumably as the result of scarring produced by the ulcer. Another alteration may be that of elongation of the pyloric canal. This may result from the narrowing of the immediate prepyloric region due to spasm, scarring or possibly the exaggeration of the mucosal folds in this region. The ring as a result of infiltration may become stiff and show no alteration in the size of the lumen through out prolonged observation. The most impor-

tant evidence of an ulcer at the pyloric ring is the niche, just as it is the most reliable criterion in the diagnosis of ulcer elsewhere.

Deformities Resulting from Ulcer. The presence of an ulcer may bring in its wake certain important anatomic changes which produce marked alterations in the size and shape of the stomach. Movnihan¹⁶ showed that the conception of congenital hourglass stomach was not tenable and that it was invariably secondary to organic disease. One case he reported was that of a trilobular stomach due to old ulceration with cicatricial healing at two points 3 inches apart. An excellent early monograph from a roentgen standpoint, on the subject of hourglass stomach is that of Rieder¹⁷ in which a large series of cases were subjected to surgical confirmation.

Such abnormal configuration of the stomach may be of organic or of functional origin. Even when the primary basis is organic, there is an accentuation in the degree of deformity by the added derangement of a functional nature. In organic hourglass stomach, the narrow zone of communication is greater than in the spastic variety. As a result, the two locules are separated by an elongated canal at the site of the lesser curvature. Moreover where the cause is organic due to scarring there is apt to be a superficial irregularity at the junction of both the upper and the lower locules. A niche is not uncommonly seen at the lesser curvature border at the origin of the biloculation.

As the area of communication becomes increasingly narrowed, there is partial obstruction to the flow of barium from the upper into the lower locule. The barium may then be noted passing slowly through this narrowed region and dropping to the lower pole of the stomach. The primary impression may be that of a stomach of which the distal portion has been altered by a malignant infiltration. Prolonged, careful observation will finally demonstrate the true nature of the lesion.

An important point in the roentgen diag-

a great tendency for occlusion with gastric retention. This may be the result of either spasm or actual cicatrization and in many cases is probably due to a combination of both factors. It is important to remember, however, that spasm may be a significant factor in producing secondary gastric enlargement and abnormal retention. This is particularly important to remember when a decision must be made as to whether medical or surgical management should be undertaken in a given case, since a marked degree of gastric retention at 24 hours or longer is ordinarily considered a point in favor of surgical interference. A knowledge, therefore, of the fact that spasm at the pyloric ring may be a complicating or indeed a predominating factor in pyloric obstruction is of considerable clinical and therapeutic significance. The best means of differentiation is through an attempt at medical management, including the use of antispasmodics and of gastric lavage. The spastic factor may be eliminated thereby, and the stomach may return to normal size and function. A failure to alleviate the obstruction by this means would favor the diagnosis of actual cicatricial constriction of an unyielding character and might justify surgical intervention.

Such pyloric stenosis, however, may also result from benign lesions other than ulcer, as in cases of benign hypertrophy of the pyloric muscle, with or without a coexistent ulcer, and in some cases of carcinomatous infiltration. In rare instances an extragastric tumor may produce sufficient occlusion of the pyloric lumen to simulate the stenosis produced by an intrinsic lesion.

One of the earliest roentgen manifestations of pyloric stenosis is an exaggeration of peristaltic activity. This may show itself in two ways: (1) the number and depth of the waves may increase, and (2) such waves may be seen originating high up in the cardiac end of the stomach. It seems almost as if the stomach, conscious of a difficulty to be overcome, made a "running start" by beginning its peristaltic activity at the

cardia. In the early stages such increased functional activity may be able to overcome the pyloric obstruction so that no abnormal retention results therefrom. Gradually, however, the stomach may become enlarged. Evidence of gastric retention may develop. The hyperactive peristalsis may alternate with periods of quiescence. Finally, periods of atony may become a prominent part of the picture, and peristalsis may be superficial, lacking vigor and ineffective in forcing the gastric contents through the pylorus. This represents the stage of decompensation. Barium retention may be present at 24 and 48 hours and even longer. The stomach is considerably enlarged downward and particularly to the right.

It is at this stage that the stomach at 6 hours may show the half-moon appearance so characteristic of advanced pyloric stenosis. This appearance is the result of a combination of two factors: (1) the abnormal retention, and (2) the fact that the marked enlargement to the right is as great as to the left. It is important to realize that even at this stage spasm may be an important factor, complicating and exaggerating the degree of pyloric stenosis. Rarely, antiperistalsis may be noted.

Because of the fact that the ingested barium in marked gastric enlargement may fall to the lower pole of the stomach, upward displacement of the barium manually under fluoroscopic control may be essential to delineate the remaining contour. Films in the prone position through various angles of obliquity are therefore important in order to demonstrate the integrity of the contour or reveal any evidence of irregularity which may aid in differential diagnosis. This is particularly important since, at times, malignant infiltration of the pylorus may produce the picture of pyloric obstruction seen with ulcer. Unless the entire contour of the stomach is demonstrable, differential diagnosis between the presence of a benign and a malignant lesion may be impossible. Under such conditions the only

diagnosis one may justifiably render on the basis of the roentgen findings is that of pyloric obstruction of organic origin. For all practical purposes, a 24 hour gastric retention may be accepted as definite evidence of the presence of an organic lesion.

Ulcers of the Greater Curvature
Though gastric ulcer shows a decided predilection for the lesser curvature and particularly for the anatomic pathway of the stomach known as the "Engpasse" or "Magenstrasse," occasionally an ulcer may occur elsewhere such as on the posterior wall, but only with extreme rarity does it occur on the greater curvature. Ulcerations of the greater curvature should always suggest the probability of malignancy.

Glaessner and Finsterer reported a case of ulcer of the greater curvature.

The patient was a man forty three years of age with a history of three years duration. He complained of violent pain to the left of the epigastrium which was made worse about one hour after eating and occasionally awakened him at night. He had had three gastric hemorrhages. The entire epigastric area was tender. Roentgen examination revealed a large ulcer on the greater curvature which retained the bismuth at twenty four hours when the stomach was entirely empty. At operation the greater curvature of the stomach was adherent to the spleen because of a callous ulcer which had penetrated into the lower lobe of the spleen. The ulcer was also firmly fixed to the tail of the pancreas. The ulcer was resected, and the spleen was removed. Histologic study of the ulcer revealed no evidence of malignancy. The patient made an excellent recovery.

Sutherland in his report of two cases of ulcer of the greater curvature of the stomach found only one of these lesions to be benign. In this particular case the roentgenogram showed a large niche on the greater curvature of the pars media toward which the rugal folds converged. This was verified at operation, the ulcer perforating against the sheath of the pancreas. Blaine¹⁴ recorded three cases of ulcer, one of which was operated on and the lesion found on the greater curvature.



FIG 155 Gastric ulcer

Additional cases of benign ulcer of the greater curvature confirmed by histologic study of the resected region were reported by Popovic, de Luna and Astier,¹⁵ Campbell,¹⁶ Cave,¹⁷ Russell,¹⁸ Friedman and Epstein,¹⁹ and Feld and Olivetti.²⁰

With only one possible exception ulcers of the greater curvature have in my experience, invariably proved to be malignant. In the one case in which the lesion was conceivably benign roentgen examination revealed a characteristic niche projecting from the greater curvature at the junction of the pars pylorica and the pars media. This finding was persistent fluoroscopically and roentgenographically. After medical management with amelioration of the clinical symptoms, further roentgen study

showed no evidence of this original deformity. There is, therefore, no surgical corroboration of the diagnosis.³²

Illustrative Cases J. S., male, aged 65. The patient gave a 5-month history of epigastric pain, gnawing in character, made worse by eating. He had several episodes of melena and lost 35 pounds since the onset of his illness. There was a long history of pulmonary tuberculosis. Gastric analysis showed a marked subacidity. Stool examination was persistently negative for blood.

Physical examination of the abdomen was essentially negative.

A partial gastrectomy was done for a gastric ulcer.

The pathologic diagnosis was gastric ulcer, no evidence of malignancy either in the stom-

ach or in a lymph node which was included in the specimen.

The roentgen examination (Fig. 155) showed a sharply defined niche of the lesser curvature, pars media. The lesser curvature to either side was normal. This deformity had the classic characteristics of a benign gastric ulcer. The niche was well rounded. It was located at the lesser curvature, pars media, and was free of any evidence of associated infiltration.

A somewhat larger niche is illustrated by the next case.

L. P., male, aged 51. A subtotal gastrectomy was done on the basis of a well-substantiated diagnosis of gastric ulcer. The operative findings were as follows: There was a deep ulcer



FIG 156 Gastric ulcer

on the lesser curvature of the stomach with a firm, indurated area surrounding it which felt suspiciously like carcinoma. There were no nodes in this area. There was an active ulcer in the first portion of the duodenum, with edema and scarring, and one small node in the gastrohepatic ligament.

The pathologic diagnosis was chronic gastric ulcer.

The roentgen examination (Fig 156) showed a somewhat rounded niche of the lesser curvature pars media. The lesser curvature to either side was of smooth contour. There was no evidence of infiltration. The preoperative diagnosis was gastric ulcer.

An ulcer may be present in the immediate prepyloric region.

G. H. male, aged 44. Nine years previously the patient had suffered from abdominal discomfort, relieved by food and alkalies, which was shown by roentgen examination to be due to a peptic ulcer. He remained well until 1 week before his admission to the hospital when he had a return of pain. On the evening of his admission he was seized with severe epigastric pain associated with collapse. The pain became generalized and radiated up into the chest. Physical examination revealed boardlike rigidity and rebound tenderness.

Operation revealed a perforation of a gastric ulcer at the lesser curvature about $1\frac{1}{2}$ inches proximal to the pylorus. Eight months later he returned to the hospital stating that he had been well until 3 weeks before. At this time a subtotal gastrectomy was done for a prepyloric ulcer.

The report of the macroscopic examination was: "On the lesser curvature in the prepyloric region there are two ulcers located about 1 cm apart. The larger stoma is oblong and measures about 1.5 by 0.5 cm and is the entrance to a round hemorrhagic smooth walled cavity about 1.5 cm in diameter. The lip of the cavity is undermined. This cavity contains a green pea. The other stoma is round and has a diameter of 0.7 cm and is the opening for another smooth walled cavity with a fibrous appearance and measuring about 1 cm. The tissues for 1 cm around both these areas are quite indurated and the serosa over them is fibrous. The mucosa is absent in both cavities, beginning again at their edges."

The microscopic examination showed no evidence of malignancy.

The diagnosis was chronic ulcers of the stomach.

Roentgen examination (Fig 157) revealed a niche at the lesser curvature of the stomach in the immediate prepyloric region. There was considerable shortening of the lesser curvature.

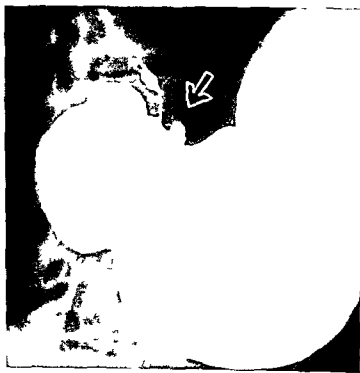


FIG 157 Prepyloric ulcer



1. The first part of the document discusses the importance of maintaining accurate records of all transactions and activities. It emphasizes the need for transparency and accountability in financial reporting.

2. The second part of the document outlines the various methods and techniques used to collect and analyze data. It includes a detailed description of the experimental procedures and the statistical analysis performed.

3. The third part of the document presents the results of the study, showing the trends and patterns observed in the data. It includes several tables and graphs to illustrate the findings.

4. The fourth part of the document discusses the implications of the results and the potential applications of the findings. It highlights the need for further research and the importance of sharing the results with the relevant community.

5. The fifth part of the document provides a conclusion and a summary of the key points discussed throughout the document. It also includes a list of references and a list of figures.

Gastrosopic examination showed a large ulcer crater on the lesser curvature just proximal to the incisura angularis measuring about $1\frac{1}{2}$ cm in diameter. There was no evidence of infiltration. Gastric analysis showed normal acidity. Examination of the gastric contents and of the stool was strongly positive for blood.

At operation, there was a large, indurated ulcer of the lesser curvature which was adherent to the pancreas. There were also some adhesions from the posterior wall of the stomach to the pancreas at the pyloric end. A partial gastrectomy was done.

The pathologic diagnosis was of a chronic gastric ulcer which had completely perforated the wall of the stomach.

Roentgen examination (Fig. 159) revealed a large niche of the lesser curvature pars media. The lesser curvature to either side of the niche was of smooth outline and somewhat retracted. There was a narrow incisura of the greater curvature opposite. The appearance

was that of a benign gastric ulcer as corroborated by microscopic examination of the resected specimen. The incisura of the greater curvature was probably of functional origin. Neither the gastrosopic examination, the surgical exploration nor the pathologic examination of the stomach after removal showed any abnormality of the greater curvature which might explain the incisura.

A wide indrawing of the greater curvature is illustrated by the next case.

K. S. female, aged 58. During the preceding 15 years the patient had had recurring episodes of right upper quadrant pain, somewhat relieved by food. The pain was worse at night. The stools were tarry on two occasions. There was moderate weight loss. A cholecystectomy and an appendectomy a number of years before admission to the hospital had failed to give relief of the pain.

Physical examination of the abdomen was essentially negative except for tenderness in



FIG. 159 Gastric ulcer with incisura of the greater curvature

the epigastrium and the right upper quadrant and the scar of the previous laparotomy

Operation revealed an ulcer high up on the posterior wall of the stomach near the lesser curvature, which was removed. In addition,

evidence of cirrhosis of the liver was found. The pathologic diagnosis was chronic ulcer of the stomach

Roentgenographic examination (Fig 160 A) showed a classical niche of the pars media

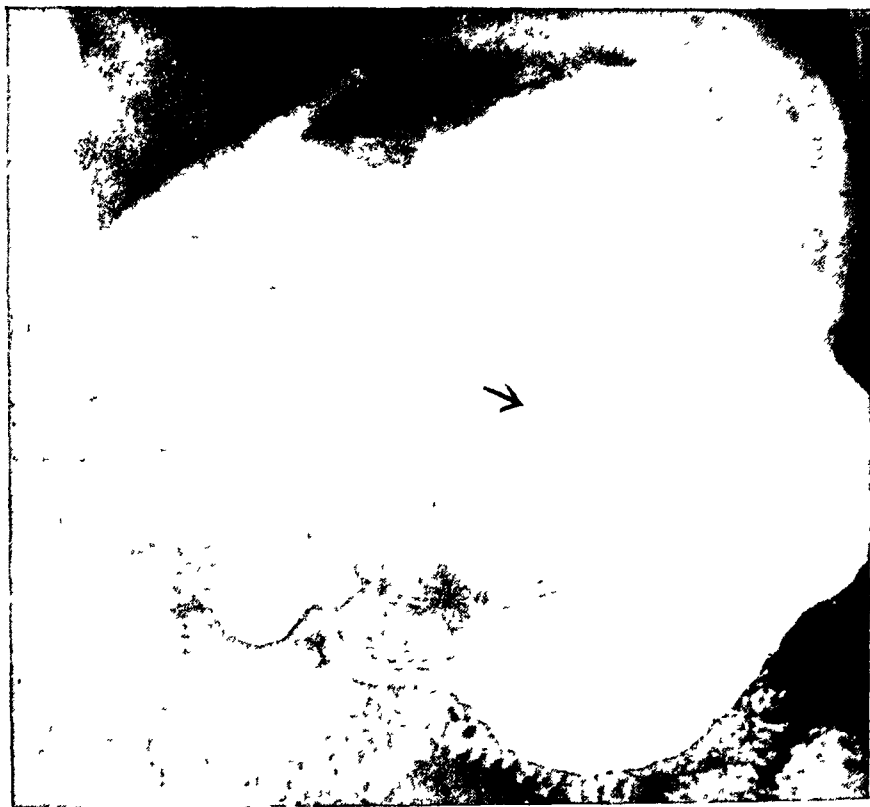


FIG 160 (A, *Top*) Gastric ulcer with incisura of the greater curvature (B, *Bottom*) Same patient. Anatomic appearance of resected ulcer



with a broad incisura of the greater curvature opposite. The anatomic appearance of the resected ulcer is shown in Figure 160 B.

The next case also illustrates a broad incisura of the greater curvature opposite a lesser curvature ulcer.

J V, male, aged 52. The patient stated that 5 years previously he had vomited a small amount of blood. Thereafter, he had recurrent attacks of blood free vomiting. During the preceding year, he had vomited daily about 2 hours after meals and during the past 4 months, he complained of a dull epigastric pain lasting from 3 to 4 hours only occasionally relieved by food. He had recently lost 20 pounds.

Physical examination of the abdomen was essentially negative except for tenderness in the mid epigastrium. The gastroscopic examination was reported as indicating a neoplasm of the posterior wall of the body of the stomach.

Roentgen examination at that time showed the presence of an ulcer of the lesser curvature, pars media of the stomach with a wide incisura of the greater curvature opposite. The surgeon at operation reported that there was an irregular nodular indurated area of the lesser curvature of the stomach to which a small amount of omentum was adherent. Small hard lymph nodes were found in the gastrohepatic omentum and scattered small nodules throughout the liver. A diagnosis of inoperable carcinoma was made and the abdomen was closed without any operative procedure on the stomach itself. No specimen was removed for biopsy.

The patient's symptoms continued unabated, with progressive weight loss. One year later, the patient was readmitted to the hospital.

Roentgen examination at this time (Fig 161) showed a large, irregularly outlined niche of the lesser curvature pars media with a wide indrawing of the greater curvature opposite. The mucosal folds appeared to be intact. Therefore it was believed that the ulcer was probably benign in spite of the fact that at the operation 1 year before the surgeon considered the lesion to be malignant.

The patient was operated upon again with the following description of the findings.

Pathology. Contrary to the previously recorded pathologic process, there was a tremendous crater defect on the posterior wall of the stomach penetrating into underlying tissue supposedly pancreas. There was a great



Fig 161 Gastric ulcer with wide incisura of the greater curvature.

deal of inflammatory reaction around the ulcer, as evidenced by edema and induration. Directly over this ulcer on the anterior wall was an indurated area in which there was some fibrosis to which the left lobe of the liver was adherent by means of an edematous band of tissue. The liver was smooth throughout, except for one portion in the left lobe which was white and scarred. This was not interpreted as neoplastic change. The stomach was not greatly dilated. The pylorus was natural. The greater omentum was adherent to the under surface of the transverse incisional scar.

A subtotal gastrectomy was done. The ulcer could not be excised from the underlying tissue and therefore the stomach was separated from it by blunt dissection leaving the crater in the underlying tissue.

The pathologic diagnosis was chronic gastric ulcer.

Autopsy 3 months after operation showed chronic fibrocaceous pulmonary tuberculosis.

Examination of the remaining portion of the stomach showed it to be anastomosed to a loop of jejunum about 18 inches from the distal end of the duodenum, widely patent and well healed. There was no evidence to indicate malignancy. The liver was normal in appearance and there were no metastatic nodes.

In spite of the report of malignancy of the

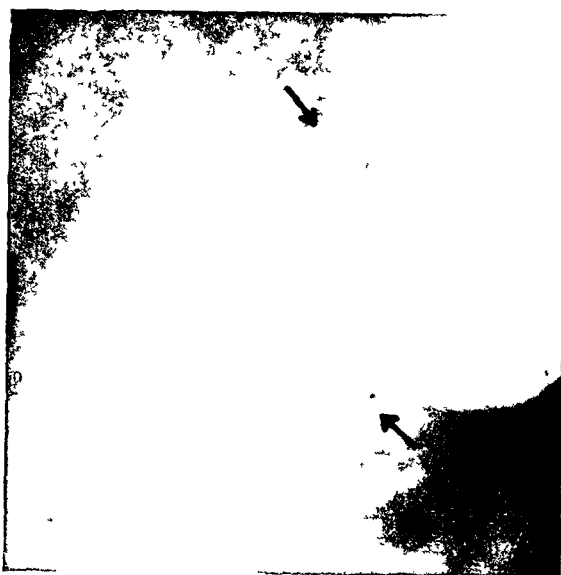


FIG 162 Organic hourglass, secondary to gastric ulcer

stomach at the first operation, the ulcer was proved ultimately to be benign. The surgical impression of the malignant nature of the lesion may sometimes require histologic confirmation.

From a study of the pathology at the site of the ulcer one may understand why the niche showed so much irregularity of contour. The ulcer was adherent to the under surface of the liver, and also the floor had penetrated to underlying tissue, apparently pancreas, and was so adherent that it could not be completely excised. Inflammation, edema and induration probably accounted for the wide incisura of the greater curvature in the region of the ulcer.

Indrawing of the greater curvature, the result of marked scarring, may ultimately lead to an actual organic hourglass stomach.

T L, female, aged 41. The patient had been complaining of recurring attacks of epigastric pain for a period of 13 years. The pain was extremely severe, radiated to the back, and occurred immediately after meals. On a careful diet she would improve for very brief periods. Attacks of pain at night waking her from sleep occurred frequently. She obtained no relief from food or soda. She became gradually worse and lost 35 pounds in weight during the course of the illness. She never vomited until shortly before her admission to the hospital, when she had a severe attack, with

coffee-colored vomitus accompanied by the passage of tarry stools.

The physical examination was negative, except for epigastric tenderness on deep palpation.

At operation, the stomach was found to be dilated, attached and held up in the center, in the shape of two separate pockets or bags, due to an ulcer of the lesser curvature. A posterior gastro-enterostomy was done, the stoma being attached to the proximal sac of the hour-glass stomach.

Roentgen examination (Fig 162) showed evidence of an ulcer of the lesser curvature, pars media. A permanent indrawing of the greater curvature separated the stomach into two locules, resulting in an organic "hour-glass" appearance.

The importance of the position of the patient in the demonstration of an organic hourglass deformity due to ulcer may be noted in the following case.

M H, female, aged 68. This patient gave a vague history of 6 years' duration of recurrent attacks of crampy abdominal pain lasting for 2 or 3 days at a time. There was occasional nausea and vomiting. She had lost 40 pounds in the preceding 6 years. Thirty-six hours before admission to the hospital she had an attack of lower abdominal pain which radiated to both shoulders and lasted about 20 minutes. On physical examination the abdomen was slightly distended and tender. At operation there were several adhesions between the liver, the lesser curvature and upper pars media of the stomach. An indurated mass was palpable on the anterior aspect of the lesser curvature, at the junction of the pars media and the pars cardiaca. Through a gastrostomy opening on the anterior wall of the stomach it was noted that the palpable mass was due to a benign ulcer. A subtotal gastric resection was done. Pathologic diagnosis: chronic ulcer of the stomach.

Radiographic examination. Figure 163 A not only shows the niche of the lesser curvature but the organic hourglass formation apparently produced by the adhesions which were found at operation. Note that this radiographic exposure was made in the erect position. Figure 163 B represents the appearance when the examination was made in the prone position. The niche cannot be seen at this time. Due also to the fact that the proximal and the distal portions of the stomach have been crowded together, there is so much

overlapping that the distinct biloculation is not demonstrable. During fluoroscopic examination, manual pressure may be necessary in order to separate the upper and the lower locules and to demonstrate clearly the intervening isthmus.

Although the niche of a benign gastric ulcer is usually of smooth, well rounded contour when completely distended with barium, there are a number of factors which may influence its appearance. One factor may be the intrusion of a blood vessel into the lumen of the ulcer. When the ulcer has perforated so that the floor is made up of pancreatic tissue, one or more translucent areas may be noted within the confines of the niche. The cause of such a transformation is illustrated by the following case.

M. D., male, aged 52. Nine months before his admission to the hospital, the patient was treated for gastric ulcer and felt well for 6 months when he had a recurrence of distress, with vomiting which contained no blood. The

distress was located over the sternum and did not radiate.

Physical examination was essentially negative, except for a right rectus scar at the site of an appendectomy.

At operation a gastric ulcer was found on the posterior wall, which had perforated through all the coats so that the body of the pancreas formed the floor of the ulcer.

A subtotal gastrectomy was done.

Examination of the resected specimen revealed the following: "On the posterior surface at the lesser curvature 8 cm proximal to the pylorus is an elliptical hole in the stomach wall 2.5 cm x 4 cm in diameter. Its edges are thickened. The mucosal surface of the specimen presents markedly hypertrophic folds localized at the cardiac half which dwindle gradually to fine small rugae at the pylorus. Microscopic examination reveals an ulcerated gastric wall with destruction of the epithelium, submucosa, muscularis and serosa. There is a marked lymphocytic infiltration on the surface of which are strands of necrotic tissue and exudate. The solitary lymph follicles are hyperplastic. The adjacent mucosa is edematous and infiltrated with lymphocytes. The mucosa adjacent to the ulcerated area is

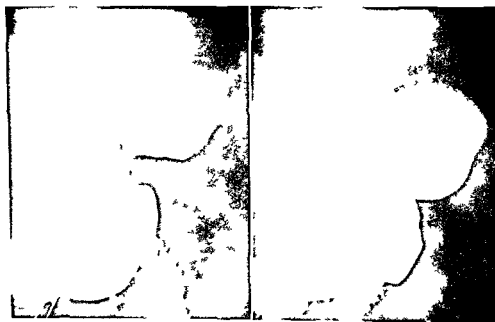


FIG. 163. Organic hourglass of the stomach secondary to an ulcer of the lesser curvature. (A) Examination in the postero-anterior erect position at 90°. Note the clear evidence of the niche and the biloculation of the stomach. (B) Examination in the postero-anterior prone position at 90°. As a result of overlapping of the proximal and distal locules of the stomach neither the niche nor the connecting isthmus between the two locules can be seen.

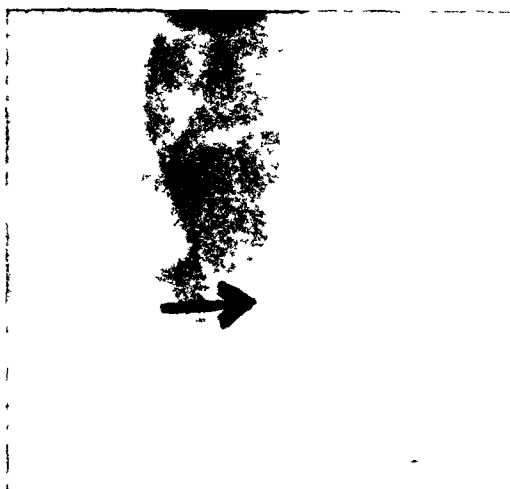


FIG 164 Gastric ulcer Note the translucent area within the niche, apparently due to the intrusion of pancreatic tissue

hyperplastic, but the muscularis mucosa in this area is intact." The diagnosis was gastric ulcer.

Roentgen examination (Fig 164) revealed a definite niche at the lesser curvature, pars media. At its apex was a rounded translucent area, which was interpreted as having been produced by a blood vessel. No such evidence

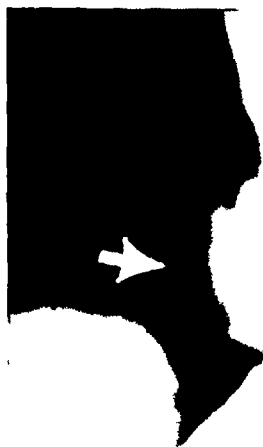


FIG 165 Shows the niche of a pathologically confirmed benign ulcer of the stomach. Within the confines of the niche are translucent areas apparently due to intrusion of the pancreas at the floor of the ulcer.

was recorded by the surgeon. In view of the fact that the ulcer had perforated through all the coats of the stomach with the pancreas forming the floor, it is possible that this translucent area at the apical portion of the niche may represent the actual intrusion of pancreatic tissue into the ulcer.

Considerable irregularity in the contour of the niche may also be noted in Figure 161, in which connection the pathologic findings were described.

Penetration of the pancreas through the floor of a gastric ulcer producing areas of translucency within the confines of the niche, is further illustrated by the following case.

L. M., male, aged 59. The patient gave a 2-year history of recurring episodes of sharp epigastric pain occurring 15 to 20 minutes after the evening meal. Frequently this was relieved by vomiting. There was a gradual increase in the severity of the pain and a loss of from 10 to 20 pounds during the year prior to hospitalization. Two weeks before admission the patient had a period of marked weakness and almost fainted. The next day his feces were "as black as coal," and his stools remained loose for about 1 to 1½ days. The vomitus never contained blood or coffee-ground material.

Physical examination was essentially negative except for deep tenderness with slight voluntary splinting in the midline just above the umbilicus. The blood count showed a marked secondary anemia. On one occasion while under observation in the hospital he vomited coffee-ground material which was found to be guaiac positive.

The report of the roentgenographic findings: "There is an ulcer of the lesser curvature, pars media of the stomach having the roentgen characteristics of a benign lesion" (Fig 165). Intraluminal translucencies within the confines of the niche were considered as being due to the intrusion of pancreatic tissue through the floor of the lesion.

A subtotal gastrectomy was performed. At operation there was a chronic gastric ulcer on the posterior wall near the lesser curvature of the pars media which had penetrated into the pancreas.

Pathologic diagnosis: chronic ulcer of the stomach.

Not only did the pathologic examination confirm the preoperative diagnosis of a benign

ulcer but it also clarified the mechanism whereby the translucent areas were formed within the niche. They evidently had resulted from the displacement of the barium within the ulcer by the pancreas into which it had eroded.

A number of other causes may produce distortion in the configuration of a niche. In the following case, F. D., male, aged 50, one may note a wavelike deformity of the outer border of the niche (Fig. 166).

Although a diagnosis of a benign gastric ulcer was made, the reason for the peculiar deformity of the contour of the niche was not clearly understood. The possibility of the indentations being due to the pressure of adherent pancreas was considered. At operation a large ulcer of the mid portion of the lesser curvature was found with chronic induration around it. This was not stuck to adjacent tissues except slightly to pancreas posteriorly. Therefore, it seemed difficult to believe that the deformity in the configuration of the niche was due to indentations produced by adherent pancreas. It is quite likely that the explanation may be found in the pathologic description of the ulcer. The pathologist reported: 'There is a small amount of fibrino purulent exudate on the surface (of the ulcer) and the base of the ulcer is composed of granulation tissue in which there is an acute and chronic inflammatory reaction. Deeper in the muscular layers, the muscle fibers are distorted by large amounts of fibrous tissue with a chronic inflammatory reaction evidenced by many lymphocytes, eosinophils and mononuclear cells. The scar tissue extends beyond the muscularis propria into the adipose tissue. Many hyperplastic lymph follicles are seen in the scar tissue and there are numerous lymph nodes in the adventitia which have hyperplastic follicles, large germinal centers and hyperplastic reticulo endothelial cells. The small arteries in the vicinity of the ulcer show marked intimal thickening and medial hypertrophy.'

Diagnosis: Chronic ulcer of stomach."

The pathologic changes in the wall of the ulcer may explain the deformity in the contour of the niche.

Irregularity in the contour of the niche may be due to a sealed off subacute perforation, as illustrated by the following case:



FIG. 166 Niche with deformity of contour in a benign chronic ulcer. Evidently the distortion of contour was due to the inflammatory changes in the floor of the ulcer described in detail by the pathologist.

J. W., male, aged 50. A few hours prior to his admission to the hospital this patient had developed a sudden attack of severe pain in the left upper abdomen radiating up into the left side of the chest to the left shoulder and down the left arm. On the evening prior to admission he had experienced paroxysmal dyspnea. The sudden onset of pain on the morning of admission was associated with a general collapse. The pain was aggravated by deep breathing. Further questioning elicited the fact that he had been having left upper quadrant pain during the preceding 2 weeks.

The physical examination was essentially negative. The clinical suspicion was that of coronary occlusion.

At operation there was found at the pars media, on the posterior wall near the lesser curvature, an area of induration with adhesions to overlying omentum. The diameter was 3 cm. A gastric ulcer was noted at this site. A posterior gastro enterostomy was done with cauterization and suture of the gastric ulcer.

Roentgen examination revealed an irregularly outlined niche with barium apparently leading from it (Fig. 167). This examination was made at the time of the patient's admis-

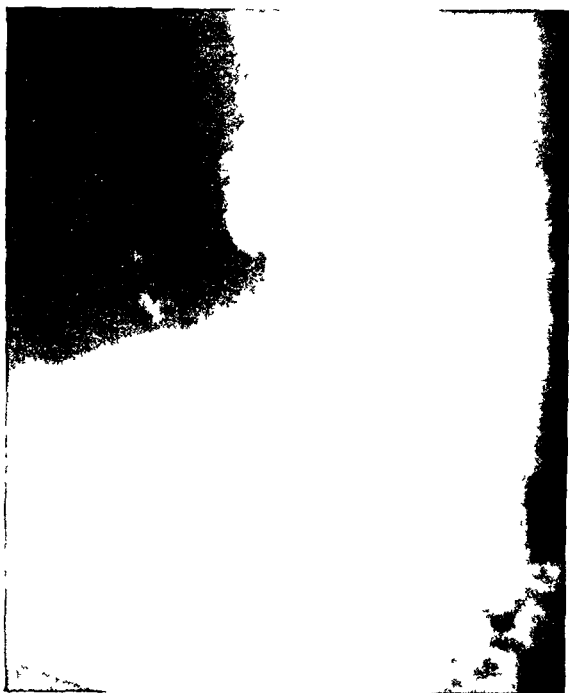


FIG 167 Gastric ulcer, showing an irregularly outlined niche, with barium apparently leading from it

sion to the hospital. The irregular outline of the niche and the apparent escape of barium from it were probably an indication of the imminence of perforation, which would correspond to the sudden onset of the left upper-

quadrant pain with collapse. Roentgen examination 3 weeks later prior to operation showed a well-rounded niche without irregularity or suspicion of escape of barium into the outside tissues. The appearance of the niche in the original examination gave the impression of a subacute perforation which had become walled off.

In some cases the only cause I have been able to discover to account for an intraluminal translucent area at the apex of the niche was a tab of omentum which had sealed off a perforation and apparently intruded within the confines of the ulcer.

The appearance of a niche will vary with the quantity of barium within the ulcer, as illustrated by the next case.

P. G., male, aged 45. This patient gave a 4 year history of upper-abdominal distress from the time of his first admission to the hospital, when he was operated on for a perforated ulcer of the duodenum. From then on he was admitted on a number of occasions because of the recurrence of epigastric pain. About 1 year after his first operation, he had a gastro-enterostomy. His most recent recurrence was of 1 month's duration, it was characterized by epigastric pain radiating to the back and not related to food or alkali intake but relieved by alcohol. He had occasional bloody



FIG 168 (A, *Left*) Gastric ulcer. Note the size of the niche when distended with barium. (B, *Right*) Same patient. Note diminished size of niche with less barium.

vomitus and tarry stools. While on the ward he had a sudden attack of severe pain in the upper abdomen which became diffuse.

Physical examination of the abdomen revealed generalized tenderness and rigidity, most marked on the right side. Operation revealed a perforated gastric ulcer at the lesser curvature.

The day before perforation the patient had been roentgenographed. Figure 168 A showed a large niche of the lesser curvature of the stomach. Figure 168 B from a picture taken during the same examination but with less barium showed the niche considerably smaller in size. The mucosal folds appeared to be unusually prominent. The diagnosis of ulcer was corroborated by the operative findings.

It is quite obvious that the size of a niche will vary with the quantity of barium in the stomach. This is one reason why, in studying the effect of any form of medical therapy upon the size of the niche, it is important that identical quantities of barium be employed. It is also clinically interesting that about 4 years previously the patient had been operated on for an acute perforation of a duodenal ulcer.

The contour of a niche will vary with the position of the patient. Thus a niche which is rounded in appearance when the patient is in the prone position (Fig 169 A) may be cone shaped in the erect position (Fig 169 B). This may be due in part to differences in the degree of distention of the ulcer with barium.

Change in the degree of obliquity even when the patient is erect may alter the appearance of the niche so that it may be cone shaped (Fig 170 A, erect posterior position at an angle of 90°) or rounded (Fig 170 B erect, first or right oblique position).

This is an important point to bear in mind in connection with the later discussion of alterations in the configuration of a niche in the healing process. Every conceivable change in the position of the patient may cause concomitant variations in the appearance of the niche. Moreover, the niche of a gastric ulcer may completely escape recognition unless the patient is examined through every angle of obliquity.



FIG 169 (A Left) Rounded niche of a gastric ulcer (B Right) Same patient. In the erect position, the niche appears cone shaped.



FIG 172 (A, *Left*) The niche of a gastric ulcer is clearly visible (B, *Right*) Same patient The niche is obscured by peristaltic waves

to a characteristic niche of the lesser curvature, corresponding to the ulcer found at operation. In Figure 172 B the arrow indicates the site where the niche should be seen, but the niche cannot be visualized. This is due to peristaltic activity, the anterior portion of the stomach near the lesser curvature bulges and temporarily conceals the more posteriorly placed niche.

The significance of the air-capped niche as evidence of chronic perforation is illustrated by the following case.

E. B., female, aged 60. The patient had been complaining for several years of pain coming on 1 hour after meals. This pain was in the epigastrium and radiated toward the left side. During the 3 weeks preceding her admission to the hospital, the pain had been constant. Occasional vomiting had occurred, which did not contain blood. She had lost 15 pounds during the preceding month.

On physical examination marked tenderness was present in the mid-epigastrium. One inch above and to the left of the umbilicus a smooth, rounded mass was palpable.



FIG 173 (*Left*) Perforating gastric ulcer with an air bubble

At operation, a very large, firm, indurated mass was present, the size of the palm of one's hand, involving the midportion of the stomach. There was a deep crater in its center. No enlarged nodes were found. Through the gastroenteric stoma the finger could be passed into a pouch extending into the lesser sac. Resection was considered inadvisable and anterior gastro-enterostomy was performed.

At autopsy, the stomach showed on its posterior wall a huge ulcer, measuring 4.5 cm in diameter, entirely ulcerated through the stomach wall and bound down by firm adhesions to the pancreas. The thickness of the ulcer was 1.5 cm. Microscopic examination of the ulcer showed no evidence of malignancy.

Roentgen examination (Fig. 173) showed a very large niche corresponding to the ulcer just described. The fluid level and surmounting gas bubble were clearly shown. That such a gas bubble was indicative of perforation was verified by the findings at autopsy, showing that all the coats of the stomach had been destroyed by the ulcer. Note also that, although the ulcer was large, pathologic examination showed the lesion to be benign.

Further evidence of the diagnostic significance of the air bubble at the upper

pole of the niche is furnished by the following case

A W, male, aged 61 The patient gave an 8 year ulcer history which had been corroborated by x ray studies The epigastric pain became steadily worse, and surgical intervention was decided upon Preoperative radiographic examination (Fig 174 A) taken in the erect position shows a large niche of the lesser curvature pars media capped by an air bubble In the prone position (Fig 174 B) the air bubble is no longer visible since it has been forced out of the ulcer Only the fairly large rounded niche is demonstrable Based on the appearance particularly in Figure 174 A showing the air bubble a diagnosis of a benign ulcer was made which had perforated and was walled off either by the pancreas or the undersurface of the liver

At operation " there was a moderate sized indurated ulcer on the middle third of the lesser curvature adherent to the pancreas posteriorly The stomach was separated from the pancreas by sharp dissection through the adherent portion which was about 2 cm in diameter A subtotal gastrectomy was done

Macroscopic Examination The muco-

exhibits a deep penetrating ulcer on the posterior side of the lesser curvature 7.5 cm from the pylorus with overhanging walls and induration of surrounding tissue It leads into the perforation seen on the serosa There is necrotic material in the crater The rest of the mucosa appears normal No lymph nodes are found in the specimen "

Microscopic diagnosis "Chronic gastric ulcer"

Thus the preoperative roentgen diagnosis as to the nature of the lesion was completely verified by the findings at operation and on pathologic examination

This case demonstrated another technical point In order to show the gas capped fluid level within the niche of an ulcer it is essential that the patient be examined in the erect position as noted in Figure 174 A

An additional example of the diagnostic significance of the air bubble in relation to the niche is illustrated by this case

B B male aged 50 The patient gave a history of recurrent attacks of pain in the left upper quadrant during a period of 8 years The pain was crampy radiated to the back and frequently woke him from sleep at night It



FIG 174 Gas capped niche of perforated gastric ulcer (A, Left) Examination in the erect position Note the gas capped niche characteristic of a benign perforating ulcer verified at operation and on pathologic examination (B, Right) Examination in the prone position fails to show the air bubble The erect position is essential for its demonstration

was relieved by food intake and medication. He lost 20 pounds in the preceding 2 months.

Physical examination was essentially negative except for tenderness in the epigastrium and the left upper quadrant.

Roentgenographic examination revealed

evidence of a large ulcer of the lesser curvature pars media (Fig 175 A and B).

In the erect position (Fig 175 A) the niche was capped by a gas bubble. In the prone position (Fig 175 B), the gas bubble was displaced by the welling up of the barium.

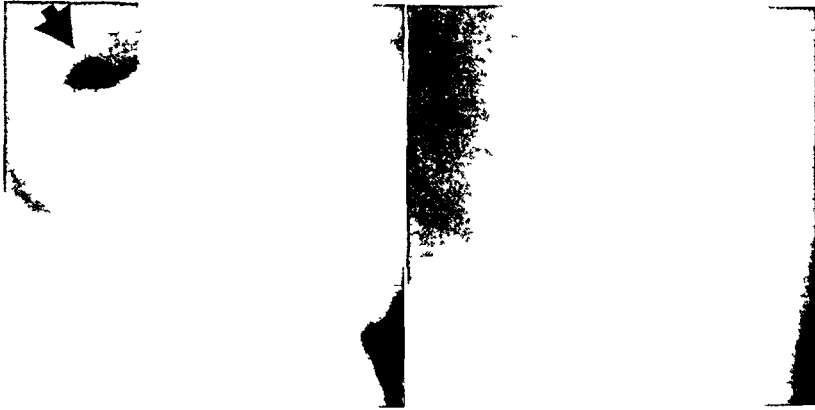


FIG 175 Air-capped niche of perforated gastric ulcer (A, *Left*) Examination in the erect position. Note the gas-capped niche. The ulcer had perforated into the pancreas. The diagnostic significance of the gas bubble as indicating a walled-off perforation of the ulcer was corroborated by operative and pathologic findings (B, *Right*) Examination in the prone position shows the distorted contour of the niche but does not show the gas bubble. Examination in the erect position is essential for the clear demonstration of the gas bubble.

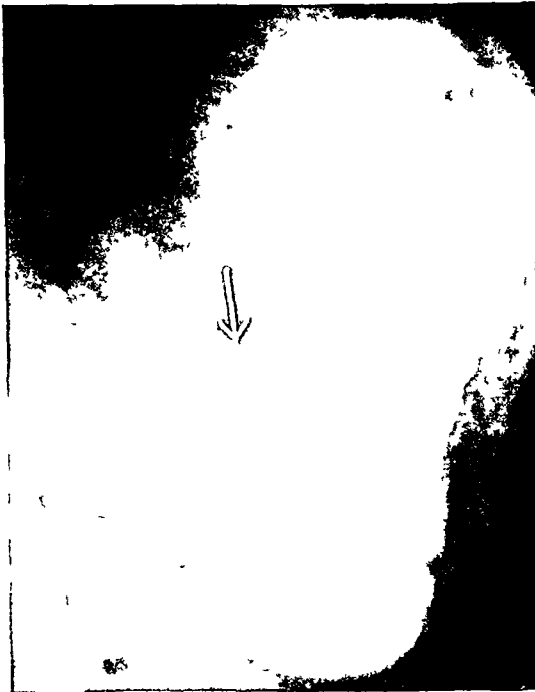


FIG 176 Air-capped niche, with shortening of the lesser curvature.

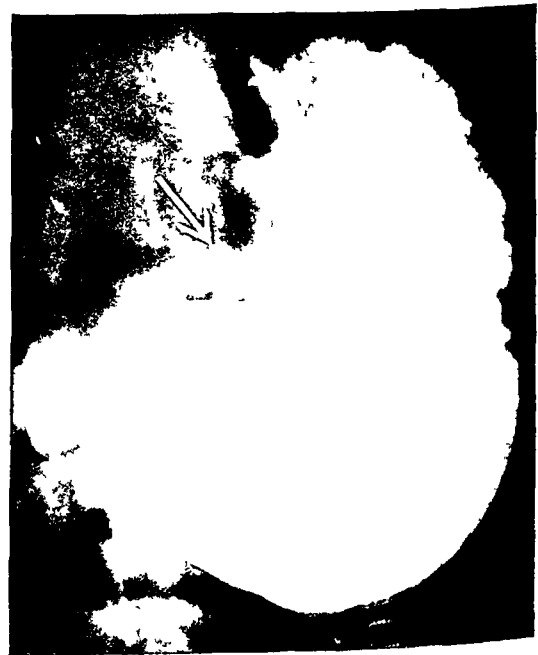


FIG 177 Marked shortening of the lesser curvature due to ulcer.

within the niche filling it completely. The contour of the niche showed a superficial irregularity. The preoperative diagnosis was of a benign ulcer that had perforated against the pancreas and was walled off by adhesions.

Operation revealed a 2 cm ulcer of the lesser curvature of the stomach pars media. The ulcer had penetrated through the wall of the stomach into the pancreas and a considerable very firm surrounding area approximately 2 cm in diameter was present. The ulcer was dissected away from the pancreas into which it had penetrated.

Macroscopic examination. On the lesser curvature and posterior aspect there is a 2.5 cm diameter excavation which extends completely through the wall of the stomach. There are attached shreds of fibrous tissue about its external periphery where it was dissected from the pancreas.

Pathologic diagnosis. "Chronic ulcer of the stomach."

The operative and the pathologic findings therefore confirmed the diagnostic significance of the air-capped niche.

Another factor of considerable importance is that of the shortening of the lesser curvature which in its extreme manifestation may produce a snail like type of stomach. The following case illustrates this.

J. J. male, aged 38. This patient complained of upper abdominal pain for a period of several years. The pain occurred from 15 to 20 minutes after food and radiated around the rib margins to the back. There was periodic remission of the symptoms. He had lost 10 pounds during the preceding year. In the 2 weeks before his admission to the hospital diet failed to relieve the pain.

Physical examination showed increased resistance over the right rectus muscle but was otherwise negative. At operation an abscess was found underneath the liver just to the right of the falciform ligament. The entire stomach was adherent and bound down to the under surface of the liver and pancreas and great difficulty was encountered in delivering the stomach into the operative wound. Partial resection was done.

Roentgen examination (Fig. 176) showed a large niche on the lesser curvature with a fluid level and an air bubble. In addition there was marked shortening of the lesser curvature of the stomach particularly of that portion distal to the niche. The pyloric arm was thereby drawn upward and toward the

proximal portion of the stomach. This shortening is due to the following factors: (1) Spasm results in functional shortening of the lesser curvature at the site of the ulcer. (2) Inflammation, with the laying down of scar tissue and its subsequent contraction leads to secondary shortening of the lesser curvature. (3) Possibly such a phenomenon may be exaggerated by the heaped up folds of mucosa at the site of the ulcer, as the result of edema and inflammation. When the shortening is of a permanent nature one may reasonably assume that this is the result of actual cicatricial inflammatory change. (4) In some cases, inflammatory involvement of the lesser omentum may cause shortening by an approximation of the lesser curvature both proximal and distal to the ulcer.

In some cases the only evidence of an ulcer may be the presence of shortening of the lesser curvature of the stomach. No niche may be demonstrable. This is illustrated by the following case.

M. S. male, aged 47. For about 16 years this patient had been complaining of periodic attacks of epigastric pain. These were not definitely related to meals. He vomited blood and passed tarry stools. He responded clinically to ulcer treatment. The last episode began about 3 months before his hospitalization. On the day of admission he had a sudden sharp abdominal pain accompanied by bloody vomitus and tarry stool.

Physical examination of the abdomen revealed moderate abdominal distention with voluntary rigidity throughout. There was moderate tenderness in the left upper quadrant and at the umbilicus.

At operation the stomach was dilated and a markedly inflamed gastrohepatic omentum was found, which was adherent to a large ulcer involving the lesser curvature pars media causing constriction of the stomach. The gall bladder and duodenum were normal. The ulcer was thought to be too large and fixed to permit resection. A gastro-enterostomy was performed.

The most significant finding on roentgen examination (Fig. 177) was a persistent shortening of the lesser curvature at the pars media with an indrawing of the distal portion of the lesser curvature toward the proximal cardiac portion. There was an incisura along the greater curvature in the immediate prepyloric region. Apparently a factor in the shortening of the lesser curvature in this area was the markedly inflamed gastrohepatic

omentum, which was adherent to the ulcer of the stomach and which contracted it in this region. It is noteworthy that this patient showed not only a considerable gastric residue at 6 hours, but also a small amount of barium in the stomach at 24 hours.

The significance of the shortening of the lesser curvature even in the absence of a visible niche is further illustrated by the next case.

J. W., male, aged 55. The patient gave a 10-year history of recurring attacks of tarry stools and of vomiting of blood. It was because of these recurrent episodes of bleeding that the patient entered the hospital for surgical therapy. A partial gastrectomy was done.

Pathologic examination revealed an ulcer of the lesser curvature about 5 cm. proximal to the pylorus; it measured 1 cm. \times 1 cm. in diameter.

Roentgen examination (Fig. 178) revealed an area of marked shortening of the lesser curvature of the stomach at the junction of the pars pylorica and the pars media. There was no demonstrable niche. The evidence indicated the presence of an old cicatrized ulcer

of the lesser curvature at the junction of the pars pylorica and the pars media.

It is noteworthy that the last hemorrhage had occurred about 4 months before operation. The roentgen examination was made 6 weeks prior to operation. The ulcer in the interval between hemorrhage and roentgen study may very well have receded, leaving the shortened lesser curvature as the major evidence of its presence.

Of considerable roentgenologic interest is the appearance of the mucosa in relation to ulcer. In the first place, its presence in association with a gastric niche is as a rule additional evidence of the benign nature of the lesion. In some cases, the folds are parallel and show no radiation to the site of the niche. They may be unusually prominent, either as the result of edema and spasm, or, at times, as the result of an actual associated gastritis.

An example of prominent mucosal folds in association with a gastric ulcer is illustrated by the case that follows.

J. C., male, aged 56. The patient gave an 8-year history of recurring attacks of epigas-

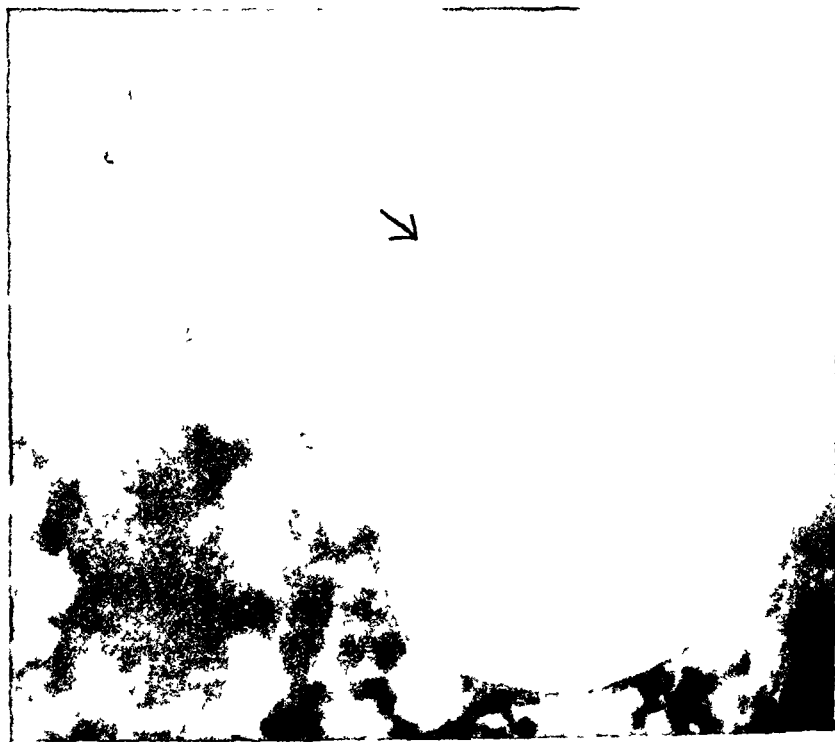


FIG. 178. Shortening of lesser curvature of the stomach due to ulcer.

tric pain after meals of vomiting and of hematemesis. The pain was relieved by the ingestion of milk and by vomiting but at the time of his admission to the hospital it was constant. Two days previously he had vomited about 1 pint of blood. He had lost 40 pounds in weight. Physical examination of the abdomen disclosed spasm throughout, most marked to the left of the epigastrium, where maximum tenderness was also present.

At operation a gastric ulcer of the lesser curvature was found adherent to the pancreas. The lesion was resected.

The pathologic diagnosis was chronic gastric ulcer.

The roentgen examination (Fig. 179) revealed a classic niche of the lesser curvature pars media. There was a fluid level within the niche capped by air. The mucosal folds of the stomach were intact. The evidence was that of a walled off perforating type of benign gastric ulcer.

Some of the folds radiate toward the niche probably as a result of puckering of the mucosa. Of considerable interest is the longitudinally placed translucent zone at the mouth of the niche representing the prominent wall surrounding the ulcer.

Examples of the radiation of mucosal folds at the site of a niche are illustrated by Figures 180, 181 and 182.

In the following case (Fig. 182) the mucosal folds are shown radiating to the

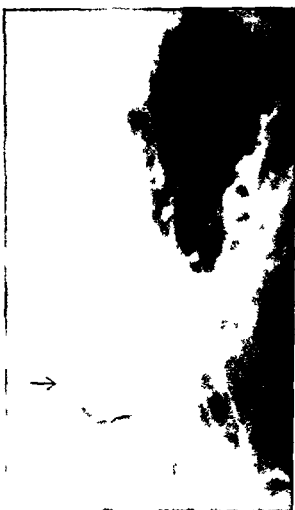


Fig. 179 The gastric mucosa in relation to ulcer



Fig. 180 (Left) Radiating mucosal folds at the site of the niche

Fig. 181 (Center) Radiating mucosal folds at the site of the niche

Fig. 182 (Right) Prominent radiating mucosal folds at the site of the niche

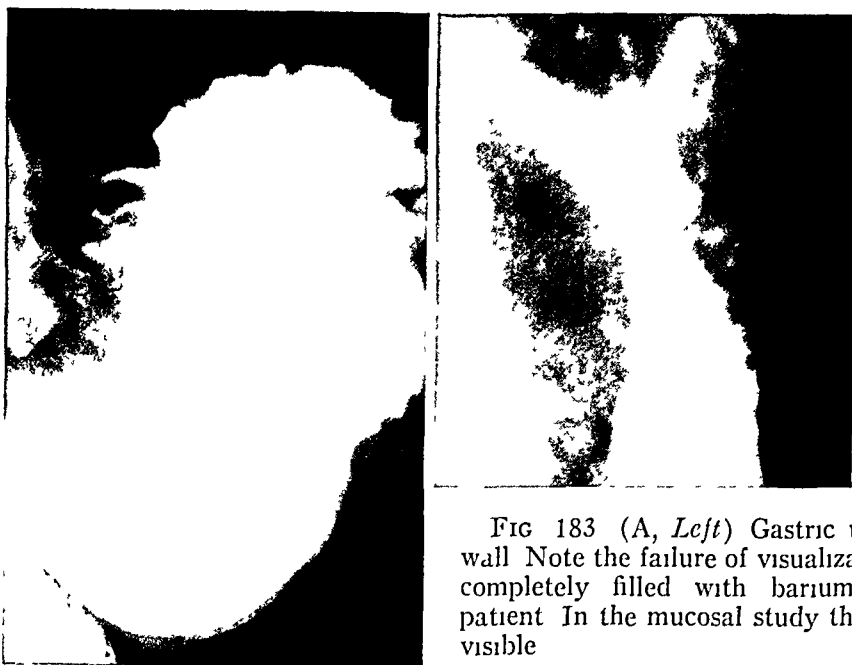


FIG 183 (A, *Left*) Gastric ulcer on the posterior wall. Note the failure of visualization with the stomach completely filled with barium (B, *Right*) Same patient. In the mucosal study the niche is now clearly visible.

site of a large niche high up in the cardiac region of the stomach. Note that the folds of the stomach appear quite prominent.

E. B., aged 46. At operation, high up on the posterior wall, near the lesser curvature of the stomach, there was a huge ulcer plastered against the pancreas. In addition, the scar of an old, healed duodenal ulcer was found. Subtotal gastrectomy was done.

The report of the macroscopic examination was as follows: "On opening the lumen the mucous membrane is found thrown up into high folds and well preserved except at the site of the defect high on the lesser curvature, where the mucous membrane is perforated by an ulcer, the base of which appears to have been left behind." The microscopic examination revealed a benign gastric ulcer.

The demonstration of an intact mucosal

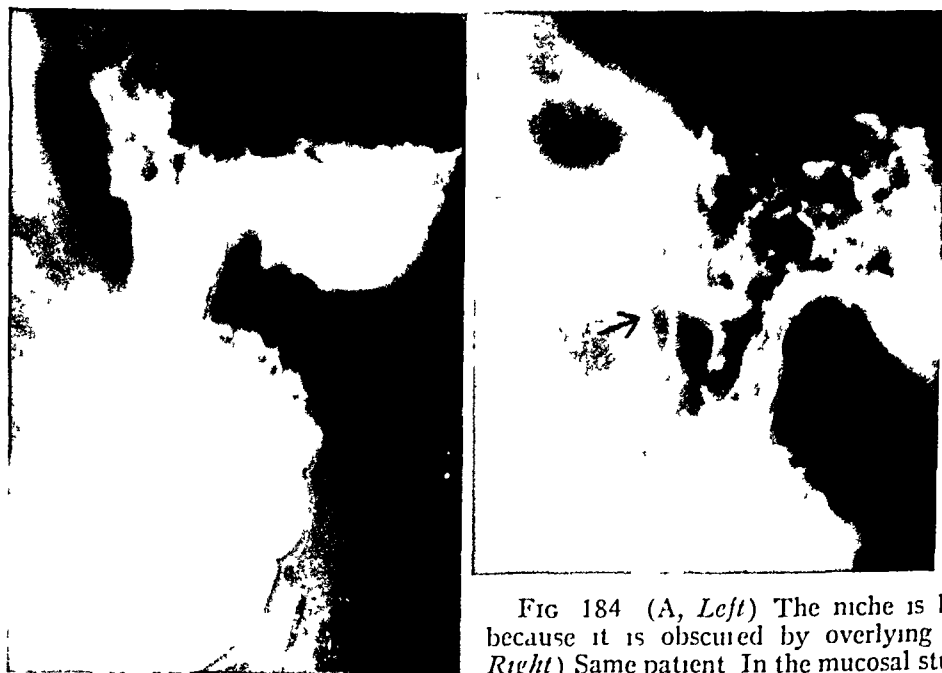


FIG 184 (A, *Left*) The niche is barely visible because it is obscured by overlying barium (B, *Right*) Same patient. In the mucosal study the niche is now clearly visible.

structure of the stomach is not only an important aid in establishing the fact that the ulcer is benign. In some cases a mucosal study may be essential in demonstrating an ulcer which may otherwise escape detection. This is particularly true when the ulcer is on the posterior wall. Such was the situation in the case next to be described.

G R, male, aged 58. The patient stated that 5 years previously he had had some stomach trouble which was diagnosed and treated as an ulcer. He remained quite well until 2 weeks prior to his admission to the hospital when he had nonradiating epigastric pain occurring 2 hours after meals. During the 4 days before admission, he vomited wine colored material and his stools were black. He felt weak, dizzy and short of breath. He had lost 30 pounds in the preceding year.

Physical examination revealed a white male markedly dyspneic and extremely pale.

Examination of the abdomen was essentially negative. Operation revealed a large gastric ulcer on the posterior wall of the stomach high up on the pars cardia. The ulcer penetrated into the tail of the pancreas. The entire cardiac end of the stomach was indurated. The ulcer was left in situ without dissecting it away from the pancreas. The ulcer itself was closed. A partial gastrectomy was done below the site of the ulcer because it was believed by the surgeon that a more radical resection would have endangered the life of the patient.

Roentgen examination (Fig 183 A) with the stomach completely filled with barium showed no evidence of any organic pathology. The mucosal relief appearance (Fig 183 B) showed a large rounded niche high up in the pars cardia corresponding to the ulcer on the posterior wall of the stomach found at operation.

It is interesting to note that even a large niche may be completely obscured when the stomach is filled with barium. This case emphasizes the importance of mucosal relief studies in the demonstration of lesions that might otherwise be obscured.

The importance of the thin layer method in showing an ulcer of the stomach which might otherwise not be visualized is illustrated by Figure 184 A and B. In Figure 184 A the niche is barely visible. In Figure 184 B the niche is clearly visible.



FIG 185 Large gastric ulcer, benign

THE SIZE OF THE ULCER AND ITS HISTOPATHOLOGY

Mere size of the niche cannot be accepted as evidence indicating the probable malignant character of the lesion. Assuming that all the other features in the roentgenogram point to the benign nature of the ulcer, a diagnosis that it is benign may be fully justified even though the niche is of large size. More important are the associated changes, such as irregularity of the lesser curvature to either side of the niche and destruction of the normal mucosal pattern. In the following case the very large niche was due to a benign ulcer.

S H, female, aged 53. During the preceding 6 weeks the patient complained of severe pain, which had begun in the lower abdomen but in a few days had become generalized finally during the preceding week becoming most marked in the umbilical region. The pain was extreme and continuous with only rare periods of relief. Vomiting occurred frequently shortly after eating. The vomitus was usually dark brown in appearance and on one occasion was very black. She had lost fifteen pounds during this period. Physical examination of the abdomen revealed generalized tenderness and a sensation of a mass in the epigastric region.



FIG 186 Large gastric ulcer, benign

Operation revealed a large, penetrating ulcer on the posterior wall of the stomach which had perforated and become adherent to the central portion of the pancreas. The area of the pancreas about this point was hard and nodular.

The pathologic diagnosis was gastric ulcer perforating into the pancreas.

The roentgen examination (Fig 185) revealed a very large niche, pars media. The contour was quite smooth except near the upper pole. The lesser curvature to either side of the niche was normal in appearance. A preoperative diagnosis of a benign ulcer was made in spite of the size of the lesion. This was based on three features: (1) its location at the pars media, (2) the essentially well-rounded configuration, and (3) the normal lesser curvature to either side of the niche, which was free of any evidence of infiltration.

An even larger niche of benign origin is illustrated by the next case.

D W, male, aged 54. The patient was perfectly well until 3 months before his admission to the hospital when he developed heartburn and epigastric pain which was aggravated by food and relieved by soda. The pain became more intense and radiated to the back. He lost 29 pounds. Gastric analysis showed a hyperacidity. While on the ward of the hospital he had a tarry stool.

Gastroscopic examination showed a large

gastric ulcer, which was reported as being probably malignant.

At operation an ulcer approximately 2.5 cm in diameter was found, situated on the posterior wall of the stomach near the lesser curvature, pars media. The ulcer was adherent to the pancreas and was surrounded by considerable dense, indurated tissue.

The macroscopic examination was as follows: "The specimen comprises about seven-eighths of the stomach. On the outer surface at the pyloric portion of the lesser curvature, the tissue is hemorrhagic and at the angle there is a perforation 1 cm x 2 cm. The tissue here is stony hard. On section this tissue appears as a ragged ulcer, with necrotic walls and thrombosed blood vessels. There are gray streaks of what appear to be tumor extending through the wall. At the pyloric region there is again hard, suspicious tissue."

The microscopic examination of the ulcer and of a perigastric node revealed no evidence of malignancy. The diagnosis was chronic gastric ulcer.

The roentgen examination (Fig 186) showed a very large, rather flat niche of the lesser curvature, pars media. It was superficially irregular. The lesser curvature just distal to the niche appeared somewhat retracted but of smooth contour. The preoperative diagnosis was ulcerating lesion, probably malignant. In this case, in spite of the size of the lesion and the irregularity of its contour, microscopic examination showed the ulcer to be benign.

It is interesting to note that the gastroscopist thought that this ulcer was probably malignant and that the pathologist in the gross examination of the specimen described the tissue as stony hard and stated that there were gray streaks of what appeared to be tumor extending through the wall and that there was hard, suspicious tissue in the pyloric region. Clinically, also, the brief history of 3 months of epigastric pain in a man of 54, with a loss of about 30 pounds in weight, would lead one to suspect a carcinoma. Microscopic study, however, ultimately proved the lesion to be benign.

The next case is that of a huge niche, with evidence of chronic perforation.

P G, male, aged 51. During the preceding year the patient complained of lower sub-



FIG. 187 (A, *Left*) Large gastric ulcer, benign. Note the small air bubble at the upper pole of the niche. (B, *Right*) Same patient. Note the appearance of the wall surrounding the niche.

sternal and epigastric pain about 15 minutes after meals. The pain radiated into the left chest to the back and on several occasions to the left shoulder. There had been occasional vomiting without blood. Nine years previously he had vomited about one half a cupful of blood. The day before being hospitalized he had had a severe hemorrhage. Physical examination of the abdomen was essentially negative.

Operation revealed a large indurated mass on the lesser curvature adherent to the pylorus. The lesion was resected. Pathologic examination disclosed a large perforation of the stomach wall on the lesser curvature.

The microscopic examination revealed chronic ulcer of the stomach.

The roentgen examination (FIG. 187 A) revealed a very large, well circumscribed niche of the lesser curvature, paracardial. The lesser curvature to either side of the niche was well defined and showed no evidence of infiltration. At the upper pole of the niche was a small accumulation of air. By means of the thin layer method the swollen wall of the ulcer could be demonstrated (FIG. 187 B). It was because of the findings that the diagnosis of a gastric ulcer was made. An ulcer which was not only benign in nature but which had perforated and become "walked off."

The presence of a fluid level capped by

air at the upper pole of the niche seen upon examination in the erect position is usually an indication that the ulcer had previously perforated. Moreover, such an appearance is strong evidence in favor of the benign nature of the lesion. I have found only one exception to this rule.

In the following case the benign character of the huge ulcer of the stomach was corroborated by autopsy.

CASE 1. Female, aged 48. The patient had been suffering from Parkinson's disease. During the preceding 6 weeks she complained of cramps in the left upper quadrant which occurred mainly at night and were relieved by the application of a hot water bottle. The pain was not related to food intake. She vomited about one hour after meals. The vomitus was black at first and later dark brown in color. The stools were occasionally black. She developed a profound secondary anemia for which she received transfusions.

Physical examination of the abdomen revealed it to be slightly distended and tympanic. There was slight tenderness on deep pressure in the left upper quadrant.

Roentgenographic examination revealed evidence of an unusually large ulcer of the stomach (FIG. 188 A).

The autopsy findings as far as the stomach is concerned were as follows "The pars media of the lesser curvature of the stomach contains a 6 x 4 5 cm ulcer, 1 cm deep with rolled, slightly indurated overhanging edges, and irregular hard floor made up of thick nodular fibrous tissue and pancreas The floor contains the mouths of several blood vessels

"Final pathologic diagnosis Chronic ulcer of the stomach with erosion of the pancreas"

Figure 188 B shows the gross appearance of the ulcer

In this case, therefore, an ulcer 6 cm x 4 5 and 1 cm deep proved to be benign

Rarely, more than one niche may be present in the stomach as a result of multiple ulcers

An example of two gastric ulcers in association with a duodenal ulcer is illustrated by the following case

C C, male, aged 62 The patient gave a 1-year history of epigastric pain, relieved by food or powders There was no vomiting or bleeding Physical examination of the abdomen was negative except for slight tenderness to the left of the epigastrium

At operation, a firm, indurated lesion was felt high up on the lesser curvature of the stomach as well as an ulcer on the posterior wall of the duodenum A partial gastrectomy was done

Pathologic examination revealed two ulcers of the stomach There was no evidence of malignancy The duodenal ulcer found at operation by the surgeon had evidently not been included in the resected specimen

The roentgen examination (Fig 189) showed a niche of the larger ulcer, high up on the lesser curvature of the stomach A smaller niche was present distally The duodenal bulb was deformed and there was a triangular-shaped niche at the border of the lesser curvature The preoperative diagnosis on the basis of this evidence was two gastric ulcers and a duodenal ulcer The surgeon at the time of operation found evidence of only one gastric ulcer and the duodenal ulcer

Pathologic examination of the specimen when opened up along the greater curvature disclosed that two gastric ulcers were present

In the following case the preoperative diagnosis of two independent benign ulcers of the stomach was also confirmed by operation and pathologic examination



FIG 188 Huge benign gastric ulcer (A, *Left*) Note the tremendous size of the niche of the lesser curvature of the stomach (B, *Right*) Appearance of the ulcer at autopsy It measured 6 x 4½ cm and 1 cm in depth



FIG 189 Two ulcers of the stomach and a duodenal ulcer

The patient J H male aged 62 gave a 20 year history of episodes of epigastric distress occurring about 2 hours after meals and occasionally awakening him from sleep at night. The pain was relieved by food and creamalin. He vomited occasionally but never any blood. There was no blood in the stools. There was no marked weight loss. Physical examination was essentially negative. Radiographic examination (Fig 190) showed the presence of two distinct niches of the lesser curvature. Upon this evidence a diagnosis of two ulcers of the stomach was made. Exploration revealed two gastric ulcers on the posterior surface of the lesser curvature, one in the midregion and one higher up. Both were adherent to the pancreas posteriorly and to the transverse mesocolon.

Macroscopic examination The stomach is opened along the greater curvature revealing two ulcers. One measures approximately 1.5 cm in the greatest diameter and is funnel shaped with thick overhanging walls. The surrounding mucosa has been replaced by scar tissue. The other ulcer is similar although only 5 mm in diameter. The pylorus is grossly normal.

Diagnosis Chronic ulcers of the stomach. Chronic gastritis.

Another example of two benign ulcers of the stomach confirmed by operation and pathologic examination is that of W J, female aged 72. Figure 191 shows the two distinct niches of the lesser curvature corresponding to two ulcers found at operation and confirmed by pathologic examination.



FIG 190 Two distinct niches representing two benign ulcers of the stomach confirmed by operation and pathologic examination

THE HEALING OF GASTRIC ULCER

That gastric ulcers frequently heal was shown by Brinton³³ many years ago. He wrote 'The cicatrix by which the ulcer heals is, on the whole, about as frequently



FIG 191 Two benign ulcers of the stomach confirmed by operation and pathologic examination

The autopsy findings as far as the stomach is concerned were as follows "The pars media of the lesser curvature of the stomach contains a 6 x 4.5 cm ulcer, 1 cm deep with rolled, slightly indurated overhanging edges, and irregular hard floor made up of thick nodular fibrous tissue and pancreas. The floor contains the mouths of several blood vessels."

"Final pathologic diagnosis: Chronic ulcer of the stomach with erosion of the pancreas."

Figure 188 B shows the gross appearance of the ulcer.

In this case, therefore, an ulcer 6 cm x 4.5 and 1 cm deep proved to be benign.

Rarely, more than one niche may be present in the stomach as a result of multiple ulcers.

An example of two gastric ulcers in association with a duodenal ulcer is illustrated by the following case.

C. C., male, aged 62. The patient gave a 1-year history of epigastric pain, relieved by food or powders. There was no vomiting or bleeding. Physical examination of the abdomen was negative except for slight tenderness to the left of the epigastrium.

At operation, a firm, indurated lesion was felt high up on the lesser curvature of the stomach as well as an ulcer on the posterior wall of the duodenum. A partial gastrectomy was done.

Pathologic examination revealed two ulcers of the stomach. There was no evidence of malignancy. The duodenal ulcer found at operation by the surgeon had evidently not been included in the resected specimen.

The roentgen examination (Fig. 189) showed a niche of the larger ulcer, high up on the lesser curvature of the stomach. A smaller niche was present distally. The duodenal bulb was deformed and there was a triangular-shaped niche at the border of the lesser curvature. The preoperative diagnosis on the basis of this evidence was two gastric ulcers and a duodenal ulcer. The surgeon at the time of operation found evidence of only one gastric ulcer and the duodenal ulcer.

Pathologic examination of the specimen when opened up along the greater curvature disclosed that two gastric ulcers were present.

In the following case the preoperative diagnosis of two independent benign ulcers of the stomach was also confirmed by operation and pathologic examination.



FIG. 188 Huge benign gastric ulcer (A, Left) Note the tremendous size of the niche of the lesser curvature of the stomach (B, Right) Appearance of the ulcer at autopsy. It measured 6 x 4½ cm and 1 cm in depth.



FIG 189 Two ulcers of the stomach and a duodenal ulcer

The patient, J H, male, aged 62 gave a 20 year history of episodes of epigastric distress occurring about 2 hours after meals and occasionally awakening him from sleep at night. The pain was relieved by food and creamalin. He vomited occasionally but never any blood. There was no blood in the stools. There was no marked weight loss. Physical examination was essentially negative. Radiographic examination (Fig 190) showed the presence of two distinct niches of the lesser curvature. Upon this evidence a diagnosis of two ulcers of the stomach was made. Exploration revealed two gastric ulcers on the posterior surface of the lesser curvature, one in the midregion and one higher up. Both were adherent to the pancreas posteriorly and to the transverse mesocolon.

Macroscopic examination. The stomach is opened along the greater curvature revealing two ulcers. One measures approximately 1.5 cm in the greatest diameter and is funnel shaped with thick overhanging walls. The surrounding mucosa has been replaced by scar tissue. The other ulcer is similar, although only 5 mm in diameter. The pylorus is grossly normal.

Diagnosis. Chronic ulcers of the stomach. Chronic gastritis.

Another example of two benign ulcers of the stomach confirmed by operation and pathologic examination is that of W J, female, aged 72. Figure 191 shows the two distinct niches of the lesser curvature corresponding to two ulcers found at operation and confirmed by pathologic examination.



FIG 190 Two distinct niches representing two benign ulcers of the stomach confirmed by operation and pathologic examination

THE HEALING OF GASTRIC ULCER

That gastric ulcers frequently heal was shown by Brinton³³ many years ago. He wrote "The cicatrix by which the ulcer heals is, on the whole, about as frequently



FIG 191 Two benign ulcers of the stomach confirmed by operation and pathologic examination

appearance of the niche. This is due to the fact that such failure to visualize a previously observed niche may result from occlusion of the ulcer with mucus, a blood clot or food or from rapid diminution of the swelling of the mucosal folds surrounding the ulcer, with a corresponding diminution in depth, or from occlusion of the neck of the ulcer by the swollen mucosa so that barium cannot enter. An excellent check on the healing process may thus be furnished by the gastroscopist, who can actually observe by direct inspection all phases in the transition from an active lesion to its complete disappearance. In this manner, Schindler, in one case, showed actual correspondence between the epithelization of the ulcer and the disappearance of the niche in the roentgenogram. In other cases, however, the niche disappears much more rapidly than the ulcer itself and is not visible roentgenologically at a time when the gastroscopist finds the healing process

to be incomplete. The average time for complete healing of an ulcer under effective treatment, according to Schindler, is 7½ weeks, although in individual cases the duration may be more protracted. Even enormous ulcers may heal with complete epithelization and no visible scar.

An important point regarding the characteristics of the niche of a gastric ulcer is its contour. Ordinarily rounded it may show irregularities at its base because of concomitant anatomic deformities. An interesting feature is the occasional funnel shaped appearance. This is sometimes noted during the retrogressive stage of a niche. In examining a patient and finding the niche to be funnel shaped, one may suspect that it is already in a state of retrogression. There is thus a better prognosis when the roentgen characteristics of the niche are of this funnel shaped nature. This, of course, does not preclude the possibility of enlargement of the niche after prelimi-

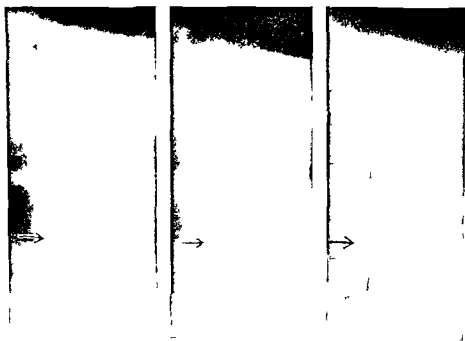


FIG. 192 (A *Left*) Niche of a gastric ulcer. (B *Center*) Same patient. The niche is smaller although the exposure was made during the same examination. (C *Right*) Same patient. The niche is still smaller here. These pictures were obtained during the course of a single examination with the patient in the same position.

nary diminution in size, with or without apparently complete disappearance. Such diminution in size has a twofold significance: (1) as a rule it means that the lesion is not of the perforating type with a rigid unyielding wall, and (2) it favors a benign nature of the lesion, although here also exceptions to the rule have been reported.

The niche of a gastric ulcer may also show an increase in size. In some cases this may indicate the imminence of perforation (Berg). This, however, is not an inevitable result of such enlargement. That an ulcer may infrequently show an increase in size has been confirmed by the gastroscopic observations of Schindler.

Illustrative Cases Evidence of moderate diminution in the size of a niche may occur even in the absence of healing. This may be due to the mucosal folds surrounding an ulcer. Transient changes in the degree of edema and swelling and spasm of the mucosal ring surrounding the ulcer may lead to concomitant changes in the apparent depth of a niche without actual healing having occurred. This is illustrated in Figure 192 A, B and C. Note that the niche appears progressively smaller in the three films, which were taken during the course of a single examination, with the patient in as

nearly an identical position as possible. Moderate diminution in the size of a niche, therefore, does not necessarily mean that there has been an actual healing of the lesion, since obviously this could not have occurred in this case.

However, that a niche may become cone-shaped in the healing process is well shown in the following case. Figure 193 A shows a moderate-sized niche of the lesser curvature, pars media. Figure 193 B shows considerable diminution in the size of the niche. It is cone-shaped in appearance at this time. Figure 193 C shows almost a complete disappearance of evidence of the original niche. However, a slight, triangular elevation at the site of the original niche is still to be noted.

At times, the niche may disappear so completely as to leave no trace of its original presence, as in the case that follows.

E. S., male, aged 52. This patient gave a 1-year history of epigastric pain, definitely relieved by milk. There was occasional vomiting, the vomitus at one time containing a small blood clot. Physical examination of the abdomen was essentially negative. The Wassermann was 4 plus. The patient did very well on medical management. Paralleling the clinical improvement, there was corresponding evidence of the favorable affect upon the ulcer in the roentgen studies. Figure 194 A showed

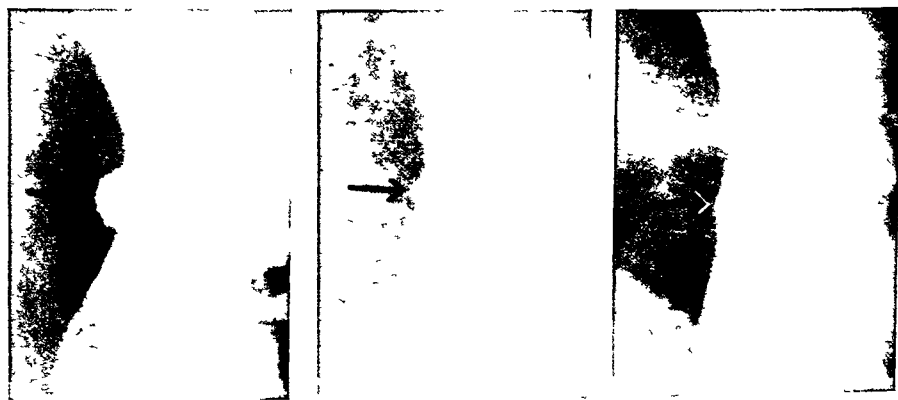


FIG 193 (A, *Left*) Niche of a gastric ulcer. (B, *Center*) Same patient. Note diminution in the size of the niche about 1 month after treatment. (C, *Right*) Same patient. Almost complete disappearance of the niche about 1 month later. A small, cone-shaped protrusion at the site of the original niche is still present.



FIG 194 (A, *Left*) Gastric ulcer (B, *Center*) Same patient 20 days later. Note the cone shaped appearance and the reduction in size (C, *Right*) Same patient 2½ months later, showing complete disappearance of the niche

the size of the niche at the first examination. Figure 194 B, 20 days later showed a reduction in the size of the niche which became triangular in shape. Figure 194 C 2½ months later showed complete disappearance of the niche.

At times in the healing process, with disappearance of the niche, a sharply defined, straight, rigid area may remain, this is

unaltered by peristaltic activity and marks the site of the original niche. The persistence of such an area of apparent stiffening may be regarded with suspicion, since recurrence may take place at this site. Such recurrence of the ulcer took place in the following case.

P B, aged 52. The patient gave a 4 year

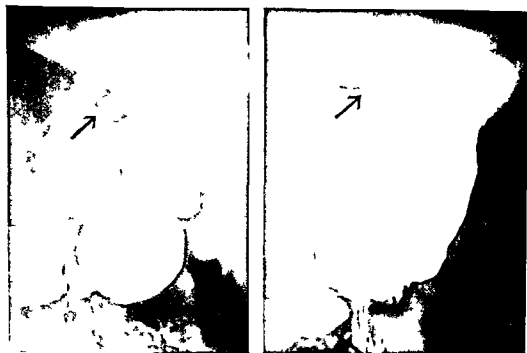


FIG 195 (A, *Left*) Niche of a gastric ulcer (B, *Right*) Same patient. There is a rigid straight line at the site of the original niche 5 months after gastroenterostomy.

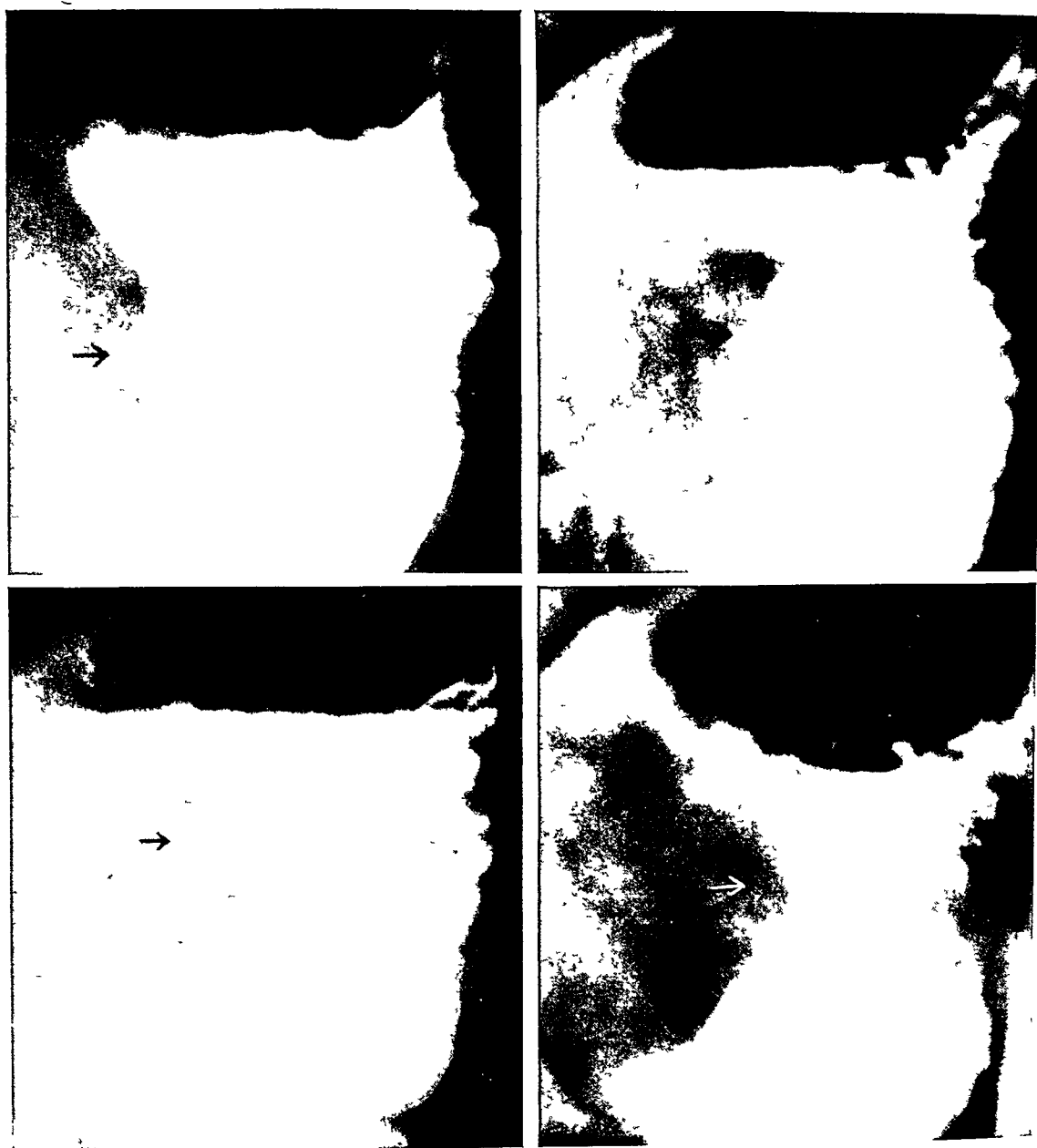


FIG 196 (A, *Top, left*) Large gastric ulcer (B, *Top, right*) Same patient Reduction in the size of the niche (C, *Bottom, left*) Same patient Still further reduction in the size of the niche (D, *Bottom, right*) Same patient Almost complete disappearance of the niche

history of epigastric pain occurring soon after meals and relieved by food. There was occasional vomiting, the vomitus was never bloody or coffee-colored. One month before admission to the hospital the patient noticed tarry stools. Physical examination was essentially negative.

Roentgen examination (Fig 195 A) revealed a niche on the lesser curvature at the

junction of the pars cardia and the pars media.

The report after operation was as follows: "Proximal to and on the posterior aspect of the pars media of the stomach there was a large indurated area about 5 cm in diameter. In the center of this area was a definite crater which admitted the tip of the little finger. The pyloric ring was patent. The ulcer-bearing

ing area seemed to be partially fixed to the posterior abdominal wall. No nodules were palpable in the liver. A gastro enterostomy was done."

Roentgen examination about 5 months later (Fig. 195 B) showed the absence of any niche formation at the site of previous involvement. A rigid sharp bar" was present at the site of the former ulcer. Later there was a recurrence of the ulcer and eventually a partial gastrectomy was performed.

The significance of a residual deformity

in the healing process is further illustrated by the next case.

T. M., male, aged 57. The patient gave a 14 month history of epigastric pain radiating to the back, occurring about 2 hours after meals and during the night and relieved by soda and food. He vomited frequently. There was no blood in the vomitus or stool. He had lost considerable weight.

Physical examination of the abdomen was negative except for epigastric tenderness. He was treated medically with a satisfactory result clinically and radiologically.

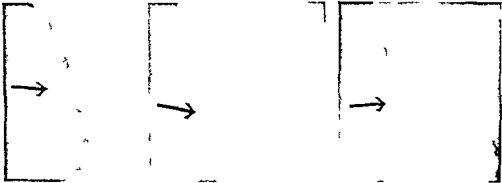


FIG. 197 (A, *Left*) Niche of a gastric ulcer (B, *Center*) Same patient. Disappearance of the niche 6 months later (C, *Right*) Same patient. Recurrence of the niche 4 months later.

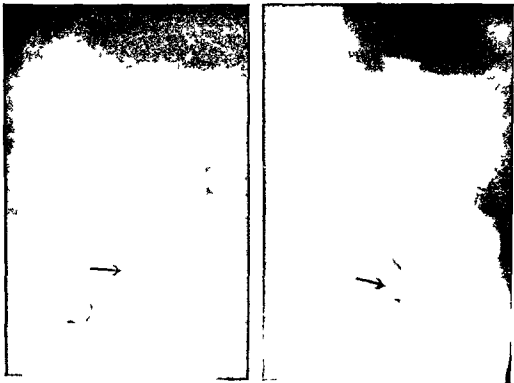


FIG. 198 (A, *Left*) Niche of a gastric ulcer (B, *Right*) Same patient. The niche shows a considerable increase in size.



FIG 199 (A, *Left*) Niche of a gastric ulcer (B, *Center*) Same patient 3 days later Note the diminution in the size of the niche (C, *Right*) Same patient 7 months later Note the marked increase in the size of the niche

Roentgen examination (Fig 196 A) showed a large niche of the lesser curvature at the junction of the pars cardia and the pars media. Figure 196 B showed a definite diminution in the size of the niche. Figure 196 C showed still further decrease in the size of the niche. Figure 196 D showed an almost complete disappearance of the niche. There was, however, a flat, cone-shaped area marking the location of the ulcer and indicating that complete healing had not taken place in spite of the dramatic diminution in the size of the niche. After remaining well for a number of months, the patient had a recurrence of intense epigastric pain and roentgen examination then showed that the niche had again become large. At operation, a large ulcer was resected, which was high up on the lesser curvature of the stomach, adherent to the pancreas. The pathologic diagnosis was gastric ulcer.

This case shows the gradual reduction in the size of the niche with almost complete disappearance but ultimately with a flare-up in the symptoms associated with a marked increase in the size of the niche. At operation a large ulcer was found.

The life cycle of the niche of a gastric ulcer is demonstrated by the following illustration. Figure 197 A shows the pres-

ence of a definite niche of a gastric ulcer at the time of clinical activity of the lesion. Figure 197 B shows the absence of any niche formation 6 months later at a time when the patient was clinically well. Figure 197 C shows a recurrence of the niche at the location of the original lesion 4 months later, which was coincidental with a recurrence of symptoms.

An increase in the size of the niche of a gastric ulcer coincident with an increase in clinical symptoms is illustrated in the following figures. Figure 198 A shows the original appearance of the gastric niche. Figure 198 B shows the appearance of the niche 1 year later, with marked increase in size. The patient had become considerably worse symptomatically. Operation finally verified the diagnosis of gastric ulcer.

In the following case there was first a reduction in the size of the niche, followed by an increase in size, so that it ultimately was larger than in the original examination.

R. D., male, aged 28. The patient gave a 6-year history of recurrent attacks of crampy pain in the epigastrium, occurring about 1 hour after meals and not relieved by food or

alkalies There had been frequent vomiting but no blood The pain sometimes awoke him from sleep Physical examination of the abdomen was essentially negative The patient was finally operated on, and an ulcer about the size of a dime was found on the lesser curvature of the stomach about $1\frac{1}{2}$ inches from the pylorus The ulcer had penetrated all the walls of the stomach and was closed by only a thin membrane and a small portion of the lesser omentum A subtotal gastrectomy was done

The report of the pathologic examination was as follows "Specimen consists of an ulcer about 1 cm \times $1\frac{1}{2}$ cm in diameter, surrounded by mucosa varying from 1 cm to 3 cm in width The whole resected specimen is elliptical in shape The ulcer is about $1\frac{1}{2}$ cm in depth and there is an opening through to the under surface The ulcer surface is somewhat hemorrhagic, and in the subserosal area there is a blood clot and a portion of the omentum appears to be adherent to the area of perforation The rim of the ulcer is firm but not stony hard No lymph node is included"

The microscopic diagnosis was chronic benign ulcer of the stomach

Roentgen examination shows interesting changes in the appearance of the niche At the time of the first examination (Fig 199 A) there was a small niche of the lesser curvature at the junction of the pars pylorica and the pars media Three days later (Fig 199 B)

examination showed a diminution in the size of the niche At this time, it was cone shaped Figure 199 C, 7 months later, showed considerable increase in the size of the niche as compared with the appearance in the preceding examination, and it was also much larger than in the original examination (Fig 199 A) The niche was sharply outlined, and the lesser curvature to either side was smooth, the entire appearance being that of a benign ulcer The gradual increase in size was apparently due to a perforation of all the layers of the wall of the stomach As noted at operation and in the pathologic examination, the perforation was walled off by a piece of omentum On further study of the niche, a slight indentation near the inferior border was noted, and the possibility suggested itself with a knowledge of the operative findings in mind, that this might represent the area of perforation sealed off by omentum

Multiple ulcers of the stomach may undergo simultaneous regression, as noted in the following case

G S, male, aged 62 This patient gave an ulcer history and roentgen examination disclosed two ulcers of the lesser curvature (Fig 200 A)

Figure 200 B about 2 weeks later, showed a reduction in the size of both niches Finally all radiologic evidence of the ulcers disappeared although the patient still complained of some epigastric distress



FIG 200 (A, Left) Two ulcers of the stomach (B, Right) Same patient two weeks later Note the diminution in the size of the two niches

RELATION OF BENIGN TO MALIGNANT GASTRIC ULCER

Apparently, the first intimation that carcinoma might arise upon the basis of a primary benign ulcer was by Cruveilhier⁴² and Rokitsky,⁴³ both of whom, however, regarded the occurrence as rare. Another important conception was that of Brinton,⁴¹ who, though he considered such transformation by no means rare, believed that it was possible for a new growth to become secondarily ulcerated in such manner as to destroy the cancer cells and leave only an apparently benign peptic ulcer in its place. Such a process might explain the presence of a lesion of this nature in association with metastatic invasion and need not necessarily imply malignant change in a primary ulcer.

Malignant transformation of an originally benign gastric ulcer is probably rare, and the unusually high figures reported by Wilson and MacCarty,⁴⁵ who stated that 68 per cent of ulcers are complicated by carcinoma, has been seriously criticized by Ewing,⁴⁶ who regarded carcinomatous transformation of a peptic ulcer as infrequent and as being probably less than the incidence of 5 per cent established by many pathologists, if only those cases are included in which there has been a long history of gastric ulcer, where the tumor is limited to isolated foci or only one portion of the ulcer, and in which the base of the lesion is free of malignant infiltration.

ROENTGEN DIAGNOSIS OF MALIGNANT GASTRIC ULCER

As a rule, the differential diagnosis of a benign from a malignant ulcer of the stomach should offer no difficulty. When the ulceration is malignant, a number of features help in its recognition. These are discussed in the following paragraphs.

1 In the presence of an ulcer of malignant character, there is apt to be an invasion of the near-by tissues, so that, roentgenologically, one may be able to

demonstrate the presence of associated irregularities of contour of the neighboring wall, with an absence of peristaltic activity in this area. Moreover, the diagnosis is simplified still further if there are irregularities of contour at some distance from the niche. When the ulcer is benign, the lesser curvature to either side is intact and shows continual alteration of contour due to the persistence of peristaltic activity. When the mucosal structure of the stomach is intact, this is in favor of the lesion's being benign.

2 The niche of a malignant ulcer may be irregular in outline and at times of unusual size. However, neither the contour nor the size are reliable criteria, the extent of ulceration may be considerable, and yet the process may be benign.

3 When the niche, even though very large, has a fluid level capped by air (in the erect position), one may consider the lesion as benign. Only with the rarest exceptions has an ulcer having this appearance ever been encountered that later proved to be malignant.

4 Another aid in differentiating a benign from a malignant lesion is the therapeutic test. After medical management has been instituted, repeated roentgen observations may be made. A diminution in the size of the niche, and particularly its complete disappearance, definitely favors the opinion that the ulcer is benign, although in rare instances this sign cannot be accepted as an absolute criterion. If, however, there is no evidence of a residuum of the niche, no matter how small, and if peristalsis through this area is normal, one may feel quite safe in eliminating malignancy as a cause of the original niche. This does not, of course, mean that if a niche fails to undergo retrogressive change under medical management it is necessarily malignant. The reason for the failure to respond to conservative treatment may be the serious inflammatory pathology of the ulcer and the hard, unyielding character of its wall. Also, a small, cone-shaped deformity of the lesser curva-

ture of the stomach as a persistent residuum in the healing process may simply mean that the ulcer, even though benign, has not undergone complete restitution and that the integrity of the wall has not been re-established. Similarly, a straight, rigid line may mark the site of the original niche and may indicate only that a weakness in the wall is present although the primary ulcerating process was benign.

In rare instances, the niche of an ulcerating lesion that ultimately proves to be malignant may apparently undergo retrogressive changes even to the point of complete and permanent disappearance of the radiologic evidence.

Rigler⁴ in 1935 described a case in which carcinomatous tissue grew into the base of the ulcer, simulating healing and another case in which the ulcer disappeared completely, although the carcinoma was increasing in size. In this connection the report by Palmer⁴⁸ is extremely interesting. He included in his paper the histologic appearance of a scar adjacent to a malignant ulcer of the stomach. This scar had become completely re-epithelized with mucosa containing nests of tumor cells. There was carcinomatous infiltration of the epithelial layer, with neoplastic glands beneath the muscularis mucosae. The major lesion, however, the malignant ulcer remained open and showed no attempt at epithelization.

Perhaps the clearest example of such roentgen evidence of the disappearance of a niche of a malignant ulcer is portrayed by Schindler and Gold.⁴¹ The roentgenogram in the first observation showed a niche of the lesser curvature having the roentgen appearance of a benign lesion. In the next observation there was a marked diminution in size and finally there was apparently complete disappearance of every vestige of the original lesion. Autopsy revealed a diffusely infiltrating carcinoma of the entire stomach with widespread metastases. An ulcer of the lesser curvature was fixed to the pancreas. The surface of the ulcer was

lined by tumor tissue, and the base showed extensive fibrosis. The accompanying photomicrograph of a section through the ulcer showed that not only had the floor of the ulcer become infiltrated with carcinoma but the gastric mucosa as well.

Mallory⁴ stated that in one case in which roentgen examination showed a diminution in the size of the niche pathologic examination of the lesion showed the former ulcer crater to be completely filled with carcinoma in situ.

Similarly, Fusterman showed the waxing and waning of a gastric niche as well as the disappearance of the ulcer and its recurrence under gastroscopic control, only to find at operation that the lesion was an ulcerating adenocarcinoma.⁴

It is hardly necessary to emphasize the fact, before concluding that evidence of an ulcerating lesion of the stomach has disappeared, that an exacting technic is essential and that reexamination should be made under the identical conditions employed when the lesion was first demonstrated. This applies not only to the position of the patient relative to the ray but also as nearly as possible to the quantity of barium with which the stomach is filled.

Mucosal studies and graded compression must also be an essential part of the technic before it is assumed that all radiologic evidence of the original lesion has disappeared. It must also be remembered that food or mucoid material or a blood clot lodged in the crater may prevent the entrance of barium for its visualization, and reexamination may be necessary particularly if there is the slightest suspicion about the findings and if they do not dovetail with the clinical manifestations. It is very probable that such a careful procedure in the corroborative examination of an ulcer patient will show that the complete disappearance of every roentgen vestige of a lesion of malignant nature is extremely rare in deed and for practical purposes, does not vitiate the clinical significance of such evi-

J. K. male, aged 60 The patient gave a 1 month history of pain in the epigastrium and in both upper quadrants The pain was sharp in character and worse after food intake Occasionally, the pain radiated to the back He had had tarry stools during the preceding week and had lost 25 pounds since the onset

Physical examination of the abdomen revealed the sensation of a mass in the left upper quadrant At operation there was a large fungating carcinoma of the distal third of the stomach There were numerous palpable nodes along the lesser curvature and several along the greater curvature There was a solitary node palpable in the liver

The report of the microscopic examination was "Specimen consists of the distal portion of the stomach unopened and unfixed measuring about 15 cm in length A large hard mass is palpable on the lesser curvature The serous coat at this point is sharply indented and there are numerous adhesions A portion of the omentum is attached and this appears fine and nodular and is infiltrated with gray tissue A small hard lymph node $1\frac{1}{2}$ cm in length is also found On opening the specimen the mucosa contains two large deep seated smooth walled ulcerations measuring

2.5 cm and 1.5 cm respectively These ulcers are situated in a thick bed of tumor tissue which extends from the mucosal surface through the entire thickness of the wall The rugal pattern of the mucosa at the margins of the growth is greatly altered The tumor extends to the pyloric ring but appears to end abruptly at that point The specimen includes the first centimeter of duodenal mucosa"

The microscopic examination revealed mucinous carcinoma of the stomach, with secondary carcinoma in the omentum and the regional lymph node

Roentgenographic examination (Fig 201) revealed a rather irregularly outlined niche at the lesser curvature, pars pylorica The lesser curvature to either side appeared rigid particularly the area distal to the niche, giving a definite impression of infiltration It was on the basis of these findings that the preoperative diagnosis was made of an ulcerating malignant lesion of the pars pylorica

The significance of rigidity of the lesser curvature of the stomach to either side of the niche is further illustrated by the next case

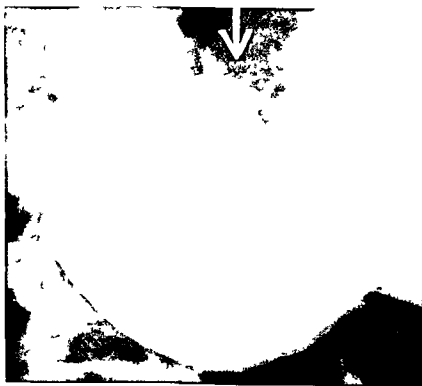


FIG 201 Malignant gastric ulcer Note the rigidity of the lesser curvature particularly in the region distal to the niche

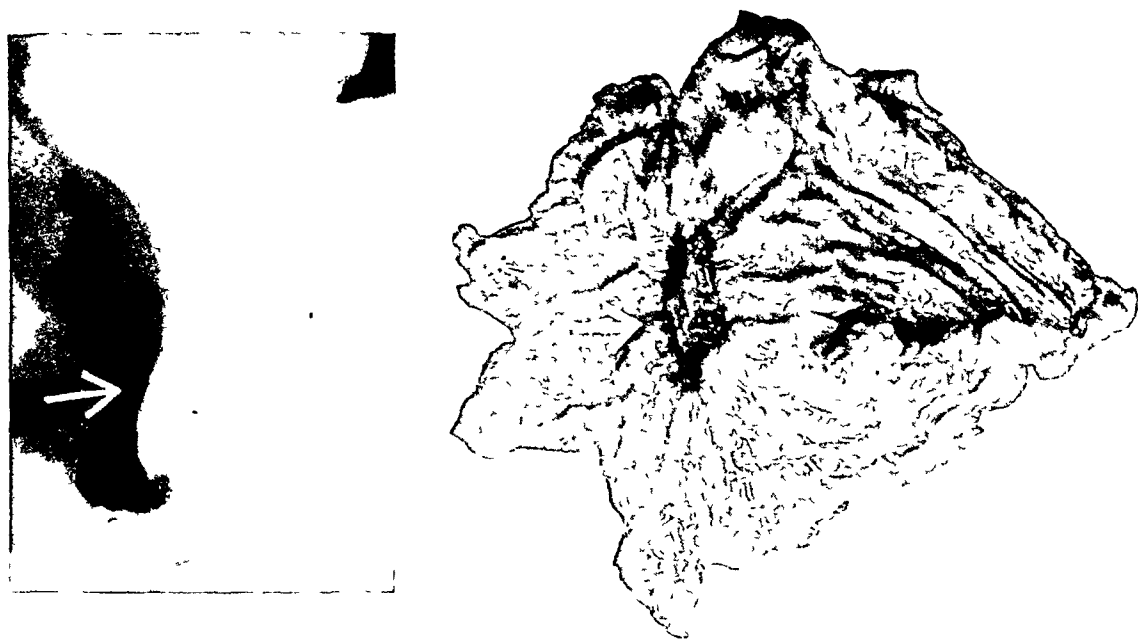


FIG 202 (A, *Left*) Malignant gastric ulcer Note that the lesser curvature to either side of the niche, is indrawn and rigid (B, *Right*) Same patient Appearance of the resected specimen

Y H, female, aged 52 The patient complained of progressive weakness for a period of 2 years, a weight loss of 34 pounds during the preceding year and epigastric pain after meals On physical examination, the liver was palpable, but abdominal examination was otherwise negative Gastric analysis showed normal acid values At operation, the surgeon stated that he found a large inflammatory

lesion at the lesser curvature of the stomach A partial gastrectomy was done

The pathologic diagnosis was ulcerative adenocarcinoma of the stomach

Roentgen examination (Fig 202 A) revealed evidence of a niche of the lesser curvature at the junction of the pars pylorica and the pars media The lesser curvature, particularly that part proximal to the niche, was indrawn and rigid The impression was that of an ulcerating lesion that had produced infiltration and stiffening of the lesser curvature proximal to it

It was because of these findings that the ulcerating lesion was believed to be malignant Figure 202 B showed the gross appearance of the resected ulcer

In the following case there was considerable distortion of the lesser curvature distal to the niche

H J, female, aged 49 The patient gave a 1-year history of occasional vomiting, weight loss of 40 pounds and epigastric soreness There was no blood in the vomitus or stool Physical examination of the abdomen was negative Gastric analysis revealed a hyperacidity

Operation revealed an ulcer about 2 inches proximal to the pylorus which almost com-



FIG 203 Malignant gastric ulcer Note the rigidity and irregularity of the lesser curvature of the stomach distal to the niche

pletely encircled the stomach. It measured nearly 1 inch in thickness in some places. The surgeon did not consider the lesion to be malignant.

The pathologic examination was carcinoma of the stomach with secondary carcinoma in a lymph node.

Roentgen examination (Fig. 203) showed a large niche of the lesser curvature, pars media. The lesser curvature distal to the niche was indrawn and rigid in appearance and did not distend in a rounded manner similar to the greater curvature opposite. The appearance was that of infiltration of the wall. These findings pointed to the malignant nature of the ulcerating lesion. As noted, the diagnosis was confirmed by the pathologist, although the surgeon at operation did not consider the lesion to be malignant.

In addition to rigidity of the lesser curvature, the fact that the niche does not project beyond it (as in the case of the typical niche already described) but remains within its confines is suggestive of ulceration within a malignant lesion. The diagnosis receives further support when the wall surrounding the niche is wide, rigid and of irregular outline. These features are illustrated by the following case.

C. M., male, aged 66. During the preceding 20 years, the patient had complained of gnawing epigastric pain occurring from 2 to 3 hours after meals and occasionally relieved by food. He remained well for a number of years, the last recurrence being 1 year before hospitalization. During this time the pain again occurred from 2 to 3 hours after meals and frequently was relieved by food or soda. The pain often occurred at night. On two occasions he noticed tarry stools lasting for 2 or 3 days. He became progressively weaker and had lost 20 pounds in the preceding year.

Physical examination revealed an extremely pale, elderly male. Examination of the abdomen disclosed an enlarged liver and the scar of a former herniotomy. There was no palpable mass.

Operation revealed a large, indurated crater on the lesser curvature of the stomach in its distal third, measuring about 5 cm. in diameter. Its surface was covered by smooth, reddened mucosa, which was not friable. There were some enlarged nodes along the greater curvature in the gastrocolic omentum. The liver was somewhat enlarged but smooth throughout. A gastric resection was done.

The report of the macroscopic examination was as follows: "On opening the specimen a large, deep ulcer is seen on the lesser curvature in the prepyloric region. Its base is smooth, dense tissue. On section, the tissue around the ulcer is denser and whiter and

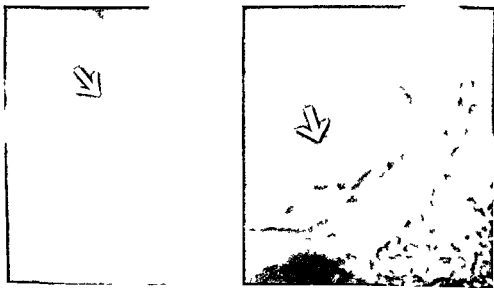


FIG. 204 (A Left) Malignant gastric ulcer. The niche is not definitely visible. (B Right) Same patient. Mucosal relief appearance showing irregularly outlined ovoid shaped niche separated from the rest of the stomach by a wide irregular translucent zone.

about 0.5 cm thick, extending almost through the wall of the stomach. The pylorus is small, stenotic and friable. No nodes are found."

The microscopic examination was reported on as follows. "Sections show that the base of the ulcer and the underlying tissue through all layers of the stomach to the serosa are made up of a malignant tumor which consists of interlacing cords of polyhedral epithelial cells which have vesicular nuclei and large conspicuous nucleoli. The edge of the ulcer shows abrupt transition into normal epithelium."

The diagnosis was undifferentiated carcinoma of the stomach.

The roentgen examination revealed the following. When the stomach was distended with barium, there was no definite evidence of any ulcerative lesion (Fig 204 A). The lesser curvature, pars pylorica, appeared rigid with a slight elevation at about its midportion. In the mucosal relief study (Fig 204 B) there was an irregularly outlined, ovoid-shaped niche, separated from the rest of the stomach by a wide, irregular, translucent zone. These findings were interpreted as representing ulceration within a new growth because of the following features: (1) the irregular configuration of the ulcerated area represented by the niche, (2) the fact that the ulcer was within the confines of the stomach and did not project outwardly beyond the contour of the lesser curvature itself, (3) the wide zone of elevation around the ulcer, representing the irregularly thickened, infiltrated wall of the new growth within which ulceration had developed.

The importance of mucosal studies as an aid in determining the malignant nature of an ulcerating gastric lesion is further illustrated by the case that follows.

A W, male, aged 55. Following the repair of a stab wound in the left upper quadrant 2½ years previously, which had nicked the gastric serosa, the patient complained of a soreness in the lower abdomen, which became a gnawing ache progressing in severity. He had lost 25 pounds. There was occasional vomiting but no blood. Physical examination of the abdomen was essentially negative. Gastroscopy revealed a necrotizing ulceration of the pylorus with regional infiltration suggestive of carcinoma.

Operation revealed a "globular multiple tumor about 1½ inches in diameter on the lesser curvature of the stomach, involving the serosa. There were several glairy mucoid nodules in the center of the mass. The gastrohepatic ligament was filled with round, hard, shotty nodes, and the dome of the left lobe of the liver and the dome of the right lobe were studded with metastatic deposits of varying sizes."

The biopsy of a nodule removed from the liver revealed metastatic adenocarcinoma.

Roentgen examination with the stomach filled with barium (Fig 205 A) showed a small area of rigidity of the lesser curvature at the junction of the pars pylorica and the pars media, without definite evidence of ulceration. The mucosal study (Fig 205 B) showed an irregularly outlined niche at the lesser curvature projecting within the lumen of the stomach. The lesser curvature, both



FIG 205 (A, Left) Malignant gastric ulcer. The niche is not clearly seen. (B, Right) Same patient. Mucosal study, showing an irregularly outlined niche at the lesser curvature, projecting within the lumen of the stomach. Note the distortion of the mucosal pattern.



FIG 206 (A, *Left*) Malignant gastric ulcer (B, *Right*) Same patient Note the importance of mucosal study in demonstrating the ulcerating lesion and the abnormality of the gastric folds

proximal and distal to the niche, appeared rigid and there was a rather wide translucent zone extending across the lumen of the stomach from the lesser toward the greater curvature. There were several additional rounded translucent areas at the base of the niche. There was also a wide defect of the greater curvature in the pars pylorica. The mucosal appearance, therefore, indicated a large ulcerating lesion extending into the lumen of the stomach and surrounded by abnormally prominent irregularly outlined gastric folds. The evidence weighed heavily in favor of an ulcerating lesion, malignant in character.

The importance of mucosal studies is further illustrated by the next case.

S. K., male, aged 49. During the preceding 6 months the patient had complained of pain in the epigastrium occurring shortly after meals. He was not relieved on an ulcer regimen. During the 4 months before his admission to the hospital he had had tarry stools and occasional vomiting. The pain finally became constant radiating to both sides and subternally. He had lost 7 pounds. Physical examination of the abdomen revealed an ill-defined mass in the epigastrium. Gastric analysis revealed essentially normal acid values.

Operation revealed a large tumor mass in the lesser curvature and the pyloric portion of the stomach. There were many pyloric nodes involved with tumor tissue. The liver was studded with small hard whitish

tumor masses ranging from $\frac{3}{4}$ mm to 1 cm in diameter. There was about 500 cc of thin yellowish fluid in the peritoneal cavity. A biopsy of the liver was taken. The pathologic diagnosis was papillary adenocarcinoma metastatic in the liver.

Roentgen examination (Fig 206 A) with the stomach distended with barium showed a small niche of the lesser curvature, pars media. From this appearance alone it would have been difficult to state with assurance that the lesion was malignant. With a thin layer of barium, however (Fig 206 B), the destruction of the mucosa was well shown. In addition there was a dense fairly well circumscribed intragastric collection of barium hugging the lesser curvature, pars media, apparently due to an ulcerating lesion. The fact that this region did not project outwardly from the lesser curvature was evidence indicating the malignant nature of the ulcerating lesion in addition to the destruction of the mucosal folds. The niche-like projection seen in Figure 206 A was not noted in Figure 206 B apparently because the barium had fallen away from this region. The preoperative diagnosis was diffuse carcinoma of the stomach.

Some important features in the differential diagnosis between a malignant and a benign ulcer of the stomach may be noted in the following case.

O. S., male, aged 55. One year and 9 months

before admission to the hospital, the patient complained of pain in the epigastrium, occurring usually at 11 A M and 4 P M, which was relieved by eating milk and crackers. After 2 weeks the pain disappeared, only to recur approximately one year later, at which time he noticed an extreme pallor. From then on he had recurring episodes of epigastric pain relieved by food. There was no vomiting, melena, or weight loss.

Physical examination of the abdomen revealed a moderately tender mass in the epigastrium. The liver was irregular and extended 4 fingers breadth below the costal margin. Gastric analysis revealed an absence of free hydrochloric acid.

In the radiographic examination of the stomach, when filled with barium (Fig 207 A), the niche exhibited the following significant features: (1) it was large in size, (2) it was of irregular contour, (3) the lesser curvature to either side of the niche was rigid, particularly in the area distal to it. This evidence of rigidity was strongly suggestive of infiltration of the wall from an invasive type of lesion. The greater curvature of the stomach, pars media, was also irregular and gave further support to the assumption of an infiltrative lesion.

The most significant findings as to the nature of the ulcerating lesion were in the mucosal pattern study (Fig 207 B). This exhibited irregularly rounded translucent areas, particularly in that portion of the stomach distal to the niche. The rest of the mucosal pattern was also devoid of normal structural characteristics. These findings were indicative of a widespread infiltrative process which had caused a destruction of the normal mucosal pattern. Therefore, the diagnosis of malignant ulcer was based primarily on the evidence of rigidity of the lesser curvature, particularly in that portion distal to the niche, as well as the abnormal mucosal pattern.

At operation there was a large hard mass involving the lesser curvature of the stomach almost up to the cardiac end, leaving only a 1-inch border of normal gastric wall on the greater curvature. The liver was completely filled with nodules of various sizes up to 5 cm. The peritoneal cavity contained a considerable amount of free fluid.

Diagnosis: Carcinoma of the stomach with metastases.

The significance of the roentgen findings in the diagnosis of malignant ulcer of the stomach therefore received complete pathologic confirmation.

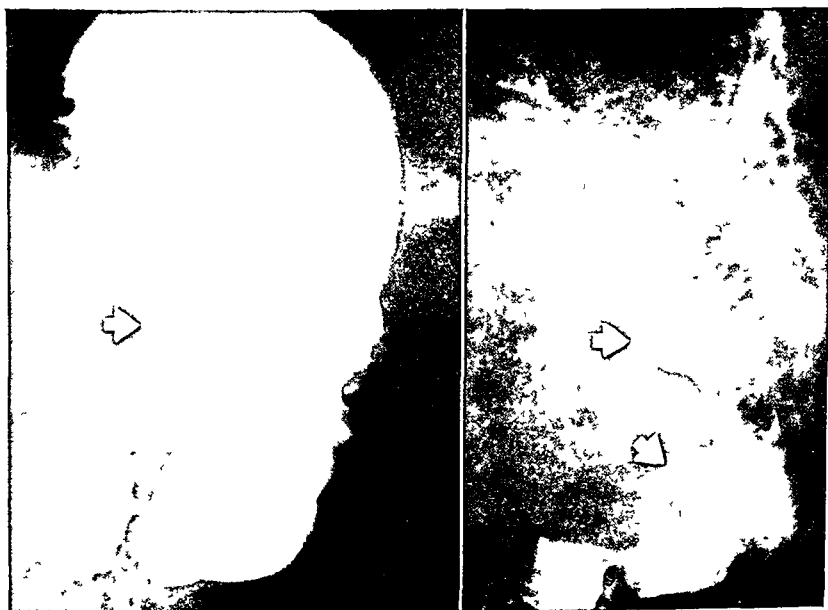


FIG 207 Malignant ulcer of the stomach (A, *Left*) Appearance of the niche in the barium-distended stomach. Note in particular rigidity of the lesser curvature distal to the niche and the irregularity of the greater curvature opposite. (B, *Right*) Mucosal pattern, showing the destruction of the normal mucosal markings produced by the malignant lesion.

Some of the technical details involved in the demonstration of a malignant ulcer, as well as the roentgen features which justify the diagnosis are illustrated by the following case

J S male, aged 58, gave a 1 year history of recurrent episodes of epigastric distress occurring about 2 hours after meals and at night. The day before hospitalization he became dizzy and vomited blood following which he fainted. His son found him in a pool of blood. His stools were black. Physical examination of the abdomen was essentially negative. The radiographic findings are of considerable interest. In Figure 208 A, the stomach being completely filled with barium, there is practically no evidence of any organic pathology. In Figure 208 B, with less barium, an ulcerating lesion may be noted which was previously obscured. The ulcerating lesion has three major characteristics: (1) it is intraluminal; (2) it is of irregular configuration; (3) it is surrounded by an irregularly outlined translucent area representing an infiltrated wall of the lesion separating it from the rest of the stomach. Based on these features a diagnosis of a malignant ulcer was made.

Operative findings: "There was on the lesser

curvature of the stomach about in its mid portion a palpable ulcerating lesion with elevated edges. Lymph nodes were palpable about the celiac axis. No nodes were noted in the omentum. No nodes were palpable or visible in the liver. The rest of the abdominal contents were negative to palpation." A total gastrectomy with esophagojejunostomy was done.

Macroscopic examination: "About 8 cm from the pyloric end on the lesser curvature there is a hard mass having a surface diameter of 5 cm, projecting into the lumen for a distance of approximately 2 cm in diameter and 1 cm in depth. A few small nodes are present in the gastrohepatic ligament."

Microscopic examination showed "a wildly growing highly anaplastic tumor of apparent mucosal origin invading all layers, including the serosa. Gland formation is frequently seen. The tumor cells are also found in cords, nests and sheets. A lymph node from the region of the celiac axis reveals invasion by tumor."

Diagnosis: "Adenocarcinoma of stomach. Secondary carcinoma in lymph nodes said to be from gastrohepatic ligament and region of celiac axis."

Therefore this case shows how easily even

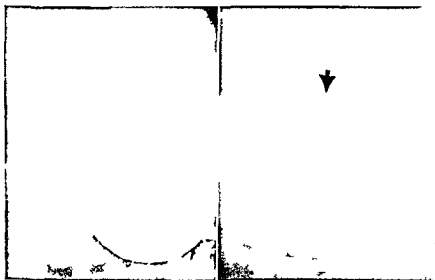


FIG 208 (A *Left*) Malignant ulcer of the stomach not visualized because the stomach is distended with barium and the lesion is obscured thereby. (B *Right*) With a thin layer of barium the roentgen features of the malignant ulcer may be seen readily. Note its intraluminal location, the irregularity of its contour, as well as the irregularly outlined translucent band separating the lesion from the rest of the stomach representing the thickened infiltrated wall about the crater.

a fairly large malignant ulcer may be obscured when the stomach is distended with barium. It also illustrates those features upon which a diagnosis of malignancy was made.

The importance of mucosal pattern compression studies of the stomach in the demonstration of a malignant ulcer may be noted in the following case.

R. B., female, aged 49. The patient gave a history of epigastric pain of 18 months' duration. At first she responded to ulcer therapy, but later the pain became intractable to any form of treatment. At one time a diagnosis of gallbladder disease was made elsewhere, and on the diet she was given she lost much weight. However, at the time of my examination, she had regained some weight. Physical examination was essentially negative.

Roentgenographic examination of the stomach when distended with barium showed no recognizable evidence of organic pathology (Fig. 209 A). During fluoroscopic examination with manual compression some deformities

of the pars media of the stomach were noted. These abnormalities were fixed in the roentgenogram by means of spot-film compression technic (Fig. 209 B). Note at this time the deformity of the lesser curvature proximal to an irregularly defined niche-like area. Note also the irregularly rounded translucent areas which characterize the mucosal relief pattern of this segment of the stomach. Based on these findings, a diagnosis of a malignant ulcerating lesion was made, and prompt surgical intervention was advised.

At operation the surgeon stated that he found a gastric ulcer and did a very extensive subtotal gastrectomy. Pathologic examination of the lesion revealed it to be a carcinoma.

Rapid increase in the size of a niche under medical observation may be suggestive of an expanding ulcerating lesion within a new growth. This is illustrated by the case that follows.

J. P., male, aged 43. The patient gave a 10-year history of episodes of indigestion

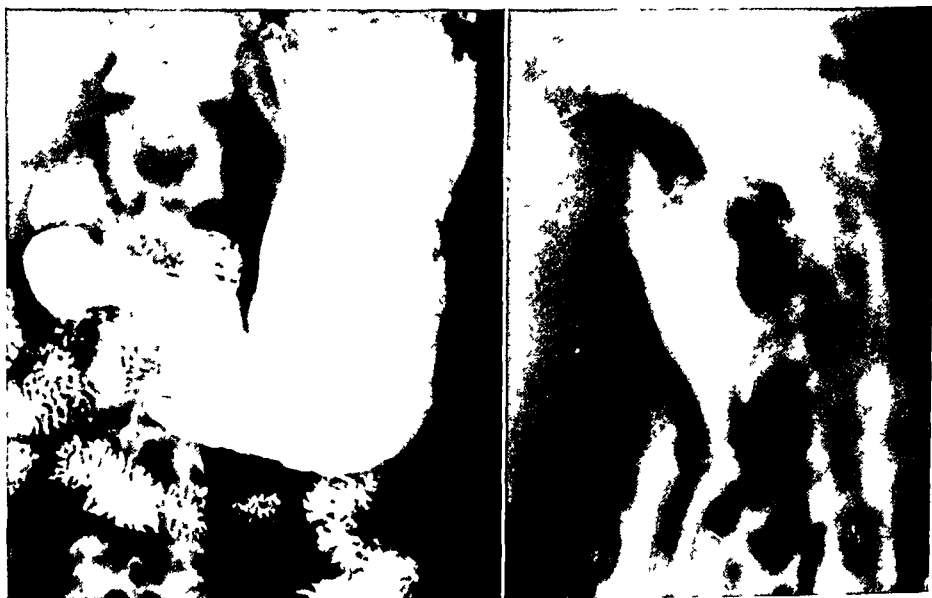


FIG. 209 (A, *Left*) Malignant ulcerating lesion of the pars media. Note the absence of any deformity at this time to indicate its presence due to the fact that the stomach is completely distended with barium. (B, *Right*) The crucial evidence of the presence of a malignant ulcerating lesion was obtained by spot-film compression technic, permitting visualization of the mucosal relief pattern. Note the deformity of the lesser curvature proximal to the irregularly outlined niche, as well as the abnormality of the mucosal pattern of that segment of the stomach characterized by rather large irregularly outlined translucent areas. These deformities were interpreted as having been produced by malignant infiltration.



FIG 210 (A Left) Malignant gastric ulcer (B Center) Same patient, 7 weeks later. Note the small, cone shaped niche (C, Right) Four weeks after B. Note the marked increase in the size of the niche.

During the preceding year, however, he had developed severe epigastric pain, occurring from 4 to 5 hours after meals, radiating to the back and relieved by food and alkalis. There was no nausea, vomiting or bleeding. The patient had gained 15 pounds. Physical examination was essentially negative except for tenderness in the epigastrium.

A subtotal gastrectomy was done, at which time the surgeon reported that there was an ulcerating lesion on the lesser curvature of the stomach. He suspected that he might be dealing with an early carcinoma. No obvious metastases were present.

The macroscopic report was "Specimen received in Bouin's solution consists of a portion of stomach which measures $10 \times 7 \times 2.5$ cm. On the lesser curvature near the pylorus is a hard, irregular nodular mass about the size of a half dollar. The rest of the serosa is smooth and glistening.

"On section, the following is revealed: there is an ulcer measuring $4 \times 3 \times 1.5$ cm on the posterior portion of the stomach near the pylorus. This ulcer has sloping edges. Its floor is ragged and nodular. The wall of the lesser curvature and of the posterior portion of the stomach in that area is replaced by dense white glistening tumor tissue. The tumor mass extends into the serosa but it is covered by it and doesn't extend further. There are no nodes present on the exterior surface of the stomach. The tumor infiltrates the entire wall and its surface is ulcerated. The fundus is uninvolved.

The microscopic report was: Sections through the stomach reveal an extensive infiltrating tumor mass. This is widely ulcerated and has penetrated through from the mucosa to the serosa and the omental fat but in all cases it is capped by the latter. The tumor

mass is, in many places, contiguous with the epithelium of the mucosa. The tumor is composed of cells of varying sizes containing one or more eccentrically placed vesicular nuclei containing one or more nucleoli. These cells are arranged in a tubular shape, for the most part. They vary in staining properties. Their growth is wild and irregular. The tumor is heavily infiltrated with acute inflammatory cells. The blood vessels show intimal proliferation and are congested and occasional hemorrhage into the tissue occurs. The fundus of the stomach is uninvolved. Along the edge of the tumor the tissue is infiltrated with chronic inflammatory cells. The tumor in no portion reaches the plane of excision. Diagnosis: Adenocarcinoma of stomach, ulcerated."

Roentgen examination showed the gradual increase in the degree of ulceration of the lesion. At first (Fig 210 A), there was a slight elevation of the lesser curvature. Seven weeks later, examination showed a small cone-shaped niche in this region (Fig 210 B). The distal lesser curvature appeared rigid. About 4 weeks later examination (Fig 210 C) showed a marked increase in the size of the niche, which was now rounded in contour. The distal lesser curvature again appeared rigid. In a period of 11 weeks there was therefore, a marked increase in the size of the niche. In addition the distal lesser curvature appeared rigid in the last two observations. Failure to operate sooner was due entirely to the patient's procrastination, operation having been recommended to him because of the increasing severity of the clinical manifestations and the exaggeration in size of the ulcerating lesion. When examined at operation this proved to be an ulcerating adenocarcinoma.

Location of a niche on the greater curvature of the stomach is strong presumptive evidence of the malignant nature of the lesion. Even in this location detailed study of the lesion may yield further evidence to substantiate the diagnosis. This is illustrated in the following case.

J. D., male, aged 62. During the preceding 3 months, the patient had complained of attacks of sharp, right upper-quadrant pain radiating subternally and toward the precordium and to the back. The pain lasted for about 1 hour and was not related to meals. He had lost 30 pounds. Physical examination of the abdomen disclosed an irregular, tender mass in the epigastrium.

Operation revealed a large ulcerating lesion of the greater curvature in the prepyloric region. A subtotal gastrectomy was done.

The pathologic diagnosis was carcinoma of the stomach.

Roentgen examination (Fig. 211 A) revealed a flat niche of the greater curvature in the pyloric portion of the stomach. The wall surrounding the niche was irregular in outline (Fig. 211 B). Apart from the fact that this lesion was on the greater curvature, which in itself would favor a diagnosis of malignancy, the roentgen characteristics were such that even if these findings were present on the lesser curvature, the diagnosis of malignant ulcer would have been justified. As an incidental finding, there was a diverticulum at the inner border of the second portion of the duodenum.

Exceptionally, a niche apparently possessing the essential characteristics of a benign lesion surmounted by an air bubble proves on histologic study to be due to an underlying malignant process. Such were the findings in the following case.

M. B., male, aged 31 years. This patient had been complaining for 2 years of pain occurring about 2 hours after meals, the pain was sharp and was relieved temporarily by soda and food. There had been periodic remissions during which he was free from distress. He had lost 48 pounds in the preceding year. His stools at times were pitch-black. Just prior to his admission to the hospital, he had had a sudden, severe attack of epigastric pain.

At the time of hospitalization, physical examination revealed generalized abdominal rigidity, with exquisite tenderness throughout, most marked in the epigastrium. There was obliteration of liver dullness.

At operation, there was a $\frac{1}{4}$ -inch perforation of an apparent gastric malignancy, which perforation was located on the anterior surface of the antrum. The malignancy extended well up on the lesser curvature. A piece of omentum was sutured at the site of the perforation.

The autopsy report was "The prepyloric region of the lesser curvature of the stomach has an ovoid ulcer about 4 cm. in diameter. It has a perforation of 1.5 cm. in diameter. This is plugged by the omentum. The floor of the ulcer has an eroded vessel."

The microscopic examination reported

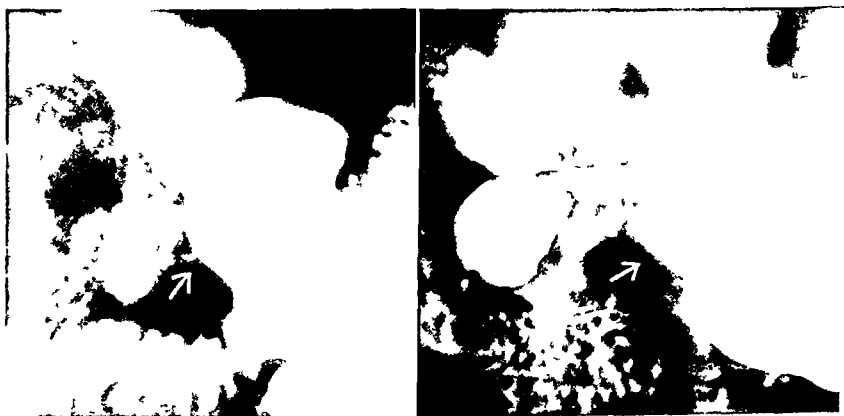


FIG. 211 (A, *Left*) Malignant ulcer of the greater curvature, pars pylorica. (B, *Right*) Same patient. The detailed characteristics of the niche are well shown. In addition, there is a diverticulum of the second portion of the duodenum.

Sections reveal a large crater in which the gastric mucosa is entirely absent with overhanging edges. The floor has four ill defined zones: (1) an inflammatory zone consisting of fibrin and polymorphonuclear leukocytes, (2) a zone of necrotic granulation tissue, (3) a zone of living granulation tissue, (4) a zone of dense scar tissue.

'In the scar tissue forming the floor of the ulcer there are large, hyperchromatic, anaplastic cells arranged in masses and in single columns separated by dense stroma. Diagnosis: Ulcerocarcinoma of the stomach. There are metastases to the nodes around the epiploic region. Sections through one of these nodes show that the normal architecture is markedly distorted. There are broad sheets of fibrous tissue invaded by large anaplastic cells with hyperchromatic nuclei. These are arranged in masses and in single columns with some glandular formations. Diagnosis: Metastatic carcinoma.'

Roentgen examination made a few days prior to the perforation (Fig 212) showed a large niche of the lesser curvature at the junction of the pars pylorica and the pars media. It was of smooth contour. It contained a fluid level line surmounted by a large air bubble. The lesser curvature of the stomach, both proximal and distal to the niche, was of



FIG 212 Gas capped niche of a malignant ulcer of the stomach (verified at autopsy)



FIG 213 (A Left) Benign gastric ulcer with roentgenographic features simulating those of a malignant ulcerating lesion. Note the defect of the lesser curvature proximal to the niche, ordinarily indicative of associated infiltration of a malignant nature, but in this case it was apparently the result of induration on an inflammatory basis. (B Right) The suspicion of malignancy in this case appeared to receive further support because of the elongated rounded translucent area in relation to the niche, which was interpreted as representing malignant infiltration of the wall of the ulcer. This, too, in retrospect was evidently due to induration of inflammatory origin.

smooth outline The diagnosis was large ulcer of the stomach, benign Because of the fluid level line and the air bubble above it, the lesion was considered to be of the perforating type Three days later, acute perforation occurred, which necessitated prompt surgical intervention The diagnosis of the malignant nature of the lesion was made at operation and on the basis of the findings at autopsy, with evidence of malignancy, not only in the ulcer itself but of metastatic invasion of a lymph node

While occasionally an ulcer having roentgen characteristics of a benign lesion may actually prove to be malignant, the reverse may also be true An ulcer which roentgenologically simulates the deformities of a malignant lesion may be found on pathologic examination to be benign

Some of the great difficulties involved in a preoperative diagnosis as to the benign or the malignant nature of an ulcer are illustrated by this case

J S, male, aged 66 The patient had always been in good health until 3 months before hospitalization He then developed abdominal pain, usually occurring about 4 hours after meals The pain was not relieved by food or medication There was no vomiting He noticed tarry stools at times He lost 30 pounds during that period The night before admission to the hospital he suddenly became unconscious This lasted for about 30 minutes On regaining consciousness he complained of generalized abdominal cramps

Physical examination of the abdomen was essentially negative There was no evidence of hemorrhage or of a cerebral vascular accident

Roentgenographic examination showed evidence of an ulcerative lesion In Figure 213 A one may note not only the niche of the lesser curvature, pars media, but also a rigid indrawing of the lesser curvature proximal to the niche Because of this evidence I con-

sidered the ulcerating lesion to be malignant This opinion was further reinforced by the evidence in Figure 213 B in which there is an elongated rather wide area of translucency in relation to the niche This was interpreted as being due to an irregular infiltrated wall surrounding the ulcer Operation, however, revealed what was apparently a benign ulcer of the pars media of the stomach on the lesser curvature extending posteriorly Examination of the liver revealed no evidence of metastases, nor was there any evidence of carcinoma elsewhere A subtotal gastrectomy was done

"The ulcerated region of the lesser curvature was found to be adherent posteriorly, and in dissecting this it was necessary to leave the base of the ulcer which was stuck down posteriorly to the base of the transverse mesocolon This left a small hole in the stomach, measuring approximately 1 cm in diameter Around the base of the ulcer which was left in the transverse mesocolon there was an area of induration about 6 cm in diameter but this did not appear to be carcinomatous "

Pathologic diagnosis Chronic ulcer of the stomach

The only way in which it seems possible to reconcile the roentgenographic findings in this case with the diagnosis of a benign ulcer is to assume that the roentgen deformities were due to the gross inflammatory pathology which transfixed the ulcer posteriorly to the base of the transverse mesocolon It is also noteworthy that there was an area of induration in the transverse mesocolon at the site of the adherent ulcer which measured about 6 cm in diameter The fixation of the ulcer to the transverse mesocolon, and apparently the wide area of induration of the tissues around it were responsible for the unusual roentgen features which so closely mimicked those found in malignant ulceration Such close simulation, in the case of a benign ulcer, of the roentgen features of a malignant ulcerating lesion as noted in this case is not common It is also noteworthy that this was a case of a benign ulcer which masqueraded as a malignant lesion and not the reverse

REFERENCES

- 1 Morgagni, J B *Recherches anatomiques sur le siége et les causes des maladies, traduites du latin par MM A Desormeaux et J P Destouet*, Paris, 1820-1824
- 2 Abercrombie, J *Contributions to the pathology of the stomach, the pancreas and the spleen, Part I, Inflammatory affections and ulceration of the stomach*, Edinburgh M & S J 21 1, 1824

- 3 Cruveilhier, J Anatomie pathologique du corps humain, ou descriptions avec figures lithographiées et coloriées des diverses alterations morbides dont le corps humain est susceptible, vol 1 Paris, Bailliere, 1829 1835
- 4 Aschoff, L Lectures on Pathology (Delivered in the United States 1924) pp 279 312, New York, Hoeber, 1924
- 5 Sturtevant N, and Shapiro L L Gastric and duodenal ulcer frequency, number, size, shape, location, color, sex and age in seven thousand seven hundred necropsy records at Bellevue Hospital, New York, Arch Int Med 38 41, 1926
- 6 Hart, G Betrachtungen über die Entstehung des peptischen Magen und Zwölffingerdarmgeschwures, Mitt Grenzgeb Med u Chir 31 350, 1919
- 7 Portis S A and Jaffe R H A study of peptic ulcer based on necropsy records, JAMA 110 6, 1938
- 8 Hemmeter J C Neue Methoden zur Diagnose des Magengeschwurs, Arch Verdauungskr 12 357, 1906
- 9 Reiche, F Zur Diagnose des Ulcus ventriculi im Röntgenbild, Fortschr Geb Röntgenstrahlen 14 171, 1909
- 10 Haudek M Zur röntgenologischen Diagnose des Ulzerationen in der Pars media des Magens München med Wchnschr 57 1587, 1910
- 11 Konjetzny, G L Die entzündliche Grundlage der typischen Geschwursbildungen im Magen und Duodenum, Berlin, Springer 1930
- 12 Frankel A Die Eigenbewegungen des Magens im Röntgenbilde Part III Fraktischdiagnostische Ergebnisse aus dem Studium der Röntgenperistaltik des Magens, Fortschr Geb Röntgenstrahlen 34 1 1926
- 13 Holmes G W and Hampton A O The incidence of carcinoma in certain chronic ulcerating lesions of the stomach JAMA 99 905 1932
- 14 Camp J D The roentgenologic significance of pyloric and prepyloric deformities Radiology 16 847 1931
- 15 Singleton A C Benign prepyloric ulcer, Radiology 26 198, 1936
- 16 Moynihan B G A Remarks on the hourglass stomach Brit M J 1 413 1904
- 17 Rieder H Die Sanduhrform des menschlichen Magens, mit besonderer Berücksichtigung der Röntgen Untersuchung, Wiesbaden, Bergmann, 1910
- 18 Schmieden, V, and Hartel F Röntgenuntersuchung chirurgischer Magenkrankheiten Berlin klin Wchnschr 46 669, 721, 772 1909
- 19 Alvarez, W C The Mechanics of the Digestive Tract ed 2, New York, Hoeber, 1928
- 20 Bauer, K H Über die Exstirpation der Magenstrasse Zentralbl Chir 48 1889, 1921
- 21 Stone R S, and Ruggles H E The diagnostic value of prepyloric and pyloric roentgen findings, Am J Roentgenol 27 193, 1932
- 22 Finsterer, H and Glaessner K In die Milz penetrierendes Ulcus der grossen Kurvature des Magens, Mitt Grenzgeb Med u Chir 27 126 1913
- 23 Sutherland, C G Niches of the greater curvature of the stomach, reports of two cases Radiology 5 248, 1925
- 24 Blaine, E S Simple penetrating ulcer of the greater curvature of the stomach Am J Roentgenol 14 20, 1925
- 25 Popovic L Eine Kasuistik (1) Ulcus curvat major in henem penetrans, etc., Fortschr Geb Röntgenstrahlen 53 337, 1936
- 26 de Luna C and Astier, A Aspect radiologique diverticulaire d'un ulcere de la grande courbure, Bull et mem Soc radiol med France 25 360, 1937
- 27 Campbell R J C Gastric ulcer of the greater curvature Brit J Radiol 21 146, 1948
- 28 Cave, P Benign ulcers of the greater curvature Brit M J 1 1185 1948
- 29 Russell W A Ulcer of greater curvature of stomach benign or malignant? Radiology 51 387 1948
- 30 Friedman R L and Epstein B S Benign gastric ulcer of the greater curvature, Radiology 55 398 1950
- 31 Feld Harold, and Olivetti R G Benign ulcer of the greater curvature of the stomach Radiology 60 53, 1953
- 32 Buckstein, J Peptic Ulcer Clinical Roentgenology with Case Histories ed 2 New York, Hoeber 1933
- 33 Brinton W Lectures on the Diseases of the Stomach, With an Introduction on Its Anatomy and Physiology, ed 2, p 240 London Churchill, 1864
- 34 Hirschfeld F Die Beziehungen zwischen Magengeschwür und Magenkrebs

- Verhandl Congr inn Med 20 279, 1910
- 35 Stewart, M J The healing of gastric ulcer, Brit M J 2 1164, 1922
 - 36 Friedenwald, J, and Baetjer, F H On the value of x-ray examinations in the diagnosis of ulcer of the stomach and duodenum, Tr A Am Physicians 28 157, 1913
 - 37 Ohnell, H Interne Behandlung bei Ulcus ventriculi mit roentgenologischer Nische, Acta med scandinav 52 1, 1919
 - 38 Hamburger, W W Roentgenological studies in the healing of gastric and duodenal ulcers, Am J M Sc 155 204, 1918
 - 39 Rosenthal, E · Über die Symptomatologie und Therapie der Magen und Duodenalgeschwüre, Berlin, Karger, 1919
 - 40 White, F W Improvement in the medical treatment of chronic ulcer of the stomach and duodenum, M Clin North America 2 1431, 1919
 - 41 Buckstein, J Roentgenographic evidence of ulcer healing, J A M A 76 231, 1921
 - 42 Cruveilhier, J Anatomie pathologique du corps humain, Paris, Baillière, 1835-1842
 - 43 Rokitsansky, C von Handbuch der speziellen pathologischen Anatomie, Wien, Braumuller and Seidel, 1842
 - 44 Brinton, W Lectures on Diseases of the Stomach, with an Introduction on Its Anatomy and Physiology, ed 2, p 240 London, Churchill, 1864
 - 45 Wilson, L B, and MacCarty, W. C. The pathological relationship of gastric ulcer and gastric carcinoma, Am J M Sc 138 847, 1909
 - 46 Ewing, J The relation of gastric ulcer to cancer, Ann Surg 67 715, 1918
 - 47 Rigler, L J The roentgen diagnosis of small carcinomata of the stomach, Wisconsin M J 34 236, 1935
 - 48 Palmer, W L Benign and malignant gastric ulcers relation and clinical differentiation, Ann Int Med 13 317, 1939
 - 49 Schindler, R, and Gold, R L Gastroscopy in gastric carcinoma, especially in its early diagnosis, Surg, Gynec & Obst. 69 1, 1939
 - 50 Mallory, T B Carcinoma in situ of the stomach and its bearing on the histogenesis of malignant ulcer, Arch Path 30 348, 1940
 - 51 Eusterman, George B Carcinomatous gastric ulcer, J A M A 118·1, 1942.
 - 52 Carman, R D Benign and malignant gastric ulcers from a roentgenologic viewpoint, Am J Roentgenol 8 695, 1921

Diverticula of the Stomach

The earliest reference to a gastric diverticulum was that by Helmont¹ Emil Hirsch in 1903 described a diverticulum of the cardia of the stomach which he had noted 2 years previously at autopsy. He commented on the fact that the egg shaped sac, of a depth of 4.5 cm., showed no scars or defects in the mucosa and that the serosa was smooth in character. Up to the time of the publication of his paper, Hirsch recorded only 14 cases of gastric diverticula in the literature including his own case.

Gastric diverticula are of comparatively rare occurrence, as shown not only in our own personal experience at Bellevue Hospital, but also in the records of other large clinics.³

There are two regions that are particularly favored in the development of gastric diverticula—the cardiac area and the pyloric end of the stomach. The site of predilection for their development is on the posterior wall of the stomach near the lesser curvature just below the level of the entrance of the esophagus. In isolated instances they have originated from the anterior wall of the stomach near the lesser curvature, pars cardia⁴ and on the greater curvature of the cardiac portion of the stomach.⁵

The wall of the diverticulum as a rule, is made up of all the layers of the stomach. In some cases the mucosa is apt to be thin and atrophic and the muscular layer poorly developed or completely absent.

The theory of the embryologic origin of diverticula at the cardia is supported by the following facts. First, the presence of diverticula during embryonic development. Thus Schwalbe⁶ described a small saccular ap-

pendage on the posterior surface of the cardia in the embryonic stomach, which he described as "diverticulum fundi" or "diverticulum ventriculi." He believed that the developmental history accounted for the persistence of such gastric diverticula in the adult. Another reason in support of the embryologic theory is the fact that such diverticula are found in the stomach of the hog and certain primates.⁷

That a gastric diverticulum may be present in the newborn is shown by Sinclair,⁸ who reported a congenital diverticulum of the stomach removed at operation in an infant 4 months old. Microscopic examination showed that it had a structure identical with that of the stomach. Symptoms necessitating operation had resulted from constriction of the pedicle by the transverse mesocolon through which the diverticulum had passed. Sir Arthur Keith in commenting on the case, stated that he entertained no doubt regarding its origin from the stomach and its congenital nature. He believed that the diverticulum originated from the dorsal border of the stomach during the second month of embryologic development.

Ogur and Kolarski⁹ reported a case of a gastric diverticulum in a newborn confirmed at autopsy. They also stated that Giles (1931) found a gastric diverticulum in a 7 year old girl. Hess and Saphir (1935) discovered a gastric diverticulum as an incidental finding in an autopsy of a 3 year old girl. Tonelli (1948) described two such cases, one in a 16 month old boy and another in a 6 1/2 month old girl.

Strong evidence is present to indicate

that, in many cases, at least diverticula at the cardia are not congenital but acquired and are the result of increased pulsion due to mechanical factors

The reason for the presence of an area of diminished resistance at the cardiac end of the stomach is explained on the basis of the following anatomic factors. At the dorsal wall of the pars cardia, there is a division of the longitudinal muscle fibers. One of these divisions passes to the greater curvature, the other passes along the lesser curvature. The circular muscle fibers, well developed at the pylorus, become structurally deficient at the cardia. The oblique fibers are also thinned out at the cardiac region of the stomach. The weakness of this area is further exaggerated by the fact that important blood vessels reach the stomach through this region. Moreover, it is precisely this area of the stomach that is subjected to considerable pressure by food entering at this point. All these factors combine in leading to the development of a herniation of the gastric tissue in this

region of anatomically diminished resistance subjected to increased intragastric pressure

Diverticula at the pylorus may also be congenital or acquired. The congenital origin of some of these diverticula is suggested by the finding of pancreatic tissue within them. Thus Falconer¹⁰ found a diverticulum arising from the greater curvature border of the pyloric canal. The diverticulum was found to contain pancreatic tissue.

Vigi and Gamberini¹¹ in the examination of a 4-year-old child at autopsy found an aberrant pancreatic nodule in the submucosal layer of a diverticulum of the stomach.

Another factor in the production of a gastric diverticulum is illustrated by Sandstrom's case.¹²

The diverticulum, irregularly outlined and narrowed in appearance, originated on the greater curvature at the pyloric end of the stomach. The roentgen diagnosis had been that of ulceration with surrounding

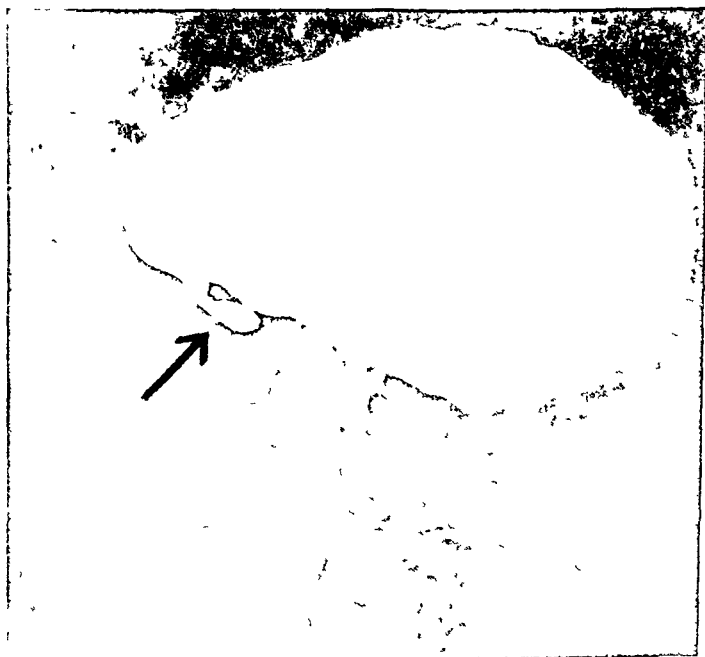


FIG. 214 Diverticulum of the stomach, showing the narrow neck

infiltration. At operation, a tumor was palpable, which was resected. When the specimen was opened it was found to consist of a diverticularlike canal lined with gastric mucosa and surrounded by a tumor like hyperplasia of glands.

ROENTGEN DIAGNOSIS

The roentgen diagnosis of a gastric diverticulum is based on the following features:

1 The location is characteristic. In the vast majority of cases, the diverticulum originates on the posterior wall near the lesser curvature, high up in the cardiac region of the stomach.

2 The diverticulum is perfectly smooth in outline, resembling in appearance diverticula elsewhere in the alimentary tract.

3 Particularly significant is the demonstration of a narrow neck of smooth delineation communicating with the stomach, commonly noted as a characteristic of diverticula generally. In rare instances the neck may be wide.

4 In the erect position the diverticulum

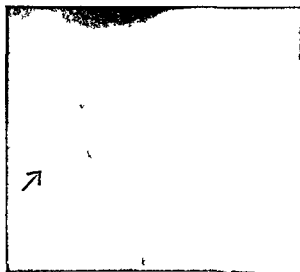


FIG. 215 Diverticulum of the stomach

will often show a fluid level capped by air, looking like a miniature gastric air bubble.

5 Strands of normal mucosa may be seen entering the diverticulum from the stomach.

6 In those cases of gastric diverticulum in which the neck is markedly narrowed, barium may be retained in the pouch at



FIG. 216 Diverticulum of the stomach showing the fluid level. Examination in the erect position.

24 hours, long after the rest of the stomach itself has emptied in a normal manner

The characteristic location and the

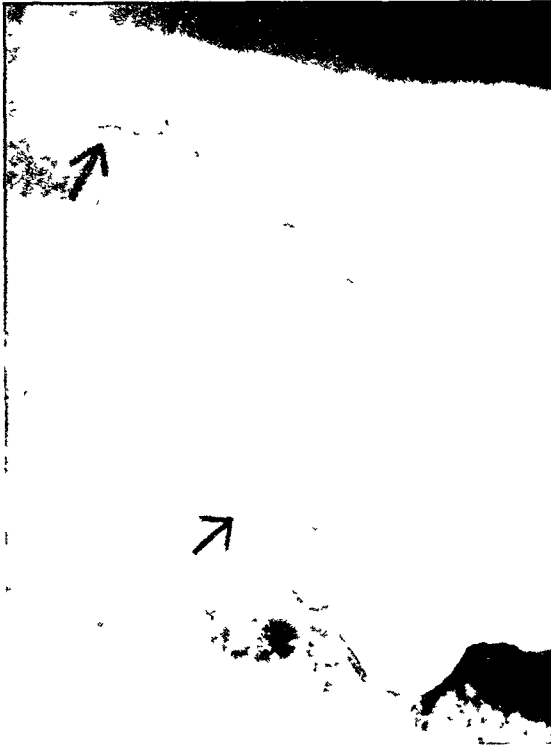


FIG 217 (A *Top*) Diverticulum of the stomach (indicated by the upper arrow) The lower arrow points to a deformity produced by a carcinoma of the pylorus (B *Bottom*) Same patient Anatomic appearance of the diverticulum



typical appearance of diverticula generally help to differentiate a diverticulum from the niche of a gastric ulcer

Illustrative Cases The following are examples of diverticula of the stomach In Figures 214 and 215, note the diverticulum originating from the usual region of the stomach, high up in the pars cardia The neck communicates between the diverticulum and the stomach

In the erect position, the smoothly outlined diverticulum high up in the cardia has a sharply outlined fluid-level line, above which is the air bubble, similar to a miniature gastric air bubble

J L, aged 50 (Fig 216) This patient's main complaint was of cough and loss of weight for a period of 7 months Roentgen examination of the lungs showed productive caseous lesions of both apices Other than the findings in the examination of the chest physical examination was essentially negative

In support of the possibility of a traumatic origin in the development of the gastric diverticulum, is the history of cough of long duration in an individual with active tuberculosis A herniation of gastric tissue may have thereby resulted at an area of the stomach of diminished resistance This explanation, however must be viewed with considerable caution, since, although pul-

monary tuberculosis is a common disease, diverticula of the stomach are comparatively rare

A diverticulum of the stomach confirmed at autopsy is described in the following case

J W, male, aged 63 During the preceding 4 months before his admission to the hospital, the patient complained of sharp pain in the epigastrium, usually after meals and some what relieved by powders No masses were palpable on abdominal examination Gastroscopic examination revealed an infiltrating lesion of the pars pylorica Some submucosal infiltrating nodules were also seen At operation, a large, hard mass was found at the lesser curvature of the stomach, pars pylorica

Pathologic examination revealed adenocarcinoma of the stomach, with metastases to the regional lymph nodes Nothing was stated about a diverticulum's being noted at the time of operation

The autopsy report was as follows "There is a diverticulum about 1.5 cm in diameter on the posterior wall of the cardiac portion of

the stomach The stoma of the gastrojejunostomy is adequate"

Roentgen examination (Fig 217 A) revealed a marked deformity of the pars pylorica, with a large defect of the greater curvature border In addition, there was a saccular area originating from the posterior wall of the pars cardia and projecting from the lesser curvature in this region because of the position of the patient (marked first oblique position) Both the carcinoma of the pylorus and a diverticulum of the pars cardia were diagnosed preoperatively The carcinoma was found at operation, but the diverticulum was overlooked at that time The autopsy, however, revealed the diverticulum on the posterior wall of the cardiac portion of the stomach Figure 217 B is a drawing of the diverticulum

The diverticulum may be outlined by air, as in the case of C Mc, which was previously described in connection with malignant gastric ulcer (See Fig 204 A and B) During the roentgen examination (Fig 218 A) it was discovered that there was a

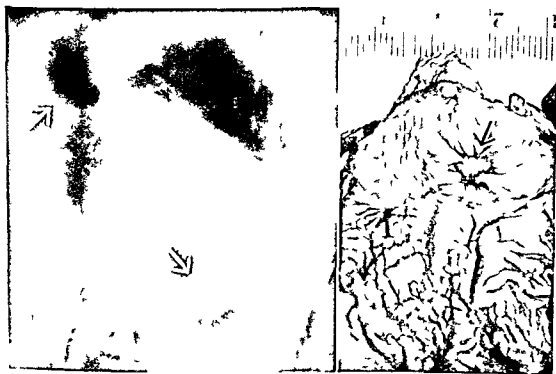


FIG 218 (A Left) Diverticulum of the stomach outlined by air (upper arrow) Note also the malignant gastric ulcer (lower arrow) which was previously described (see Fig 204 A and B) (B Right) Same patient Anatomic appearance of the diverticulum

24 hours, long after the rest of the stomach itself has emptied in a normal manner

The characteristic location and the

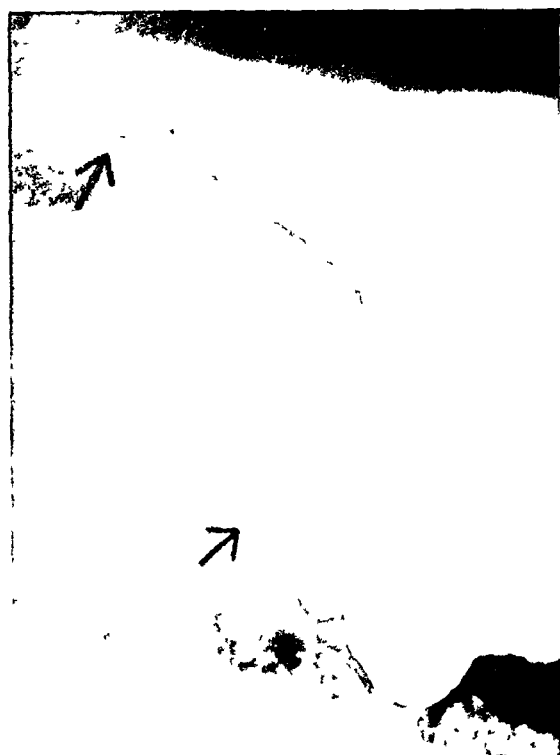


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FIG 218 (A Left) Diverticulum of the stomach outlined by air (upper arrow) Note also the malignant gastric ulcer (lower arrow) which was previously described (see Fig 204 A and B) (B Right) Same patient Anatomic appearance of the diverticulum



FIG 219 Spot film of a diverticulum of the cardiac portion of the stomach (confirmed surgically).

rounded, saccular area in the pars cardia, outlined by air, which was interpreted as being due to a diverticulum. At operation, the diverticulum was not discovered. At

autopsy, however, an opportunity presented itself of carefully studying the remaining portion of the stomach and the diverticulum was then found (Fig 218 B).

A spot film of a surgically confirmed diverticulum of the cardiac portion of the stomach is illustrated by Figure 219 (A G, female, aged 43). The patient gave a 14-year history of recurrent episodes of pain, mainly in the back and the substernal region. Occasionally, the pain woke her from sleep during the night. She stated that she felt quite comfortable, provided that she continued on an ulcer regimen. However, because of the persistent recurrences, her brother, a physician, arranged for an operation, which confirmed the diagnosis of a diverticulum on the posterior wall of the stomach. This was easily exposed and completely excised. There was no evidence of ulcer. Neither gross nor microscopic examination showed any evidence of pathology in the excised diverticulum.

REFERENCES

- 1 Quoted by Voigtel, *Handbuch der pathologische Anatomie*, p 512, 1804-1805
- 2 Hirsch, E. Über ein Magendivertikel, *Vuchow's Arch path Anat* 174 576, 1903.
- 3 Riveis, A B, Stevens, G, and Kirklin, B R. Diverticula of the stomach, *Surg, Gynec. & Obst* 60 106, 1935
- 4 Ravenel, L J. Diverticulum from the anterior surface of the stomach near the cardio-esophageal junction, *Am J Roentgenol* 10 904, 1923
- 5 Myles, R B. Anatomical variations of the stomach and duodenum within the abdominal cavity, *Brit J Radiol* 10 237, 1937
- 6 Schwalbe, G. Beiträge zur Kenntnis des menschlichen Magens, *Ztsch Morphol u Anthropol*, Sonderheft 2 1, 1912
- 7 Boppe, M. A propos du développement de l'estomac humain, *Compt rend, Association d'anatomie*, in *Bibliographie anatomique*, supp 13, pp 187-196, 1913
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Benign Tumors of the Stomach

INCIDENCE AND TYPES

The following analysis indicates the frequency of benign tumors of the stomach at Bellevue Hospital

From 1915 to 1938 there were 21 026 autopsies. Among these there were 76 cases of benign tumor of the stomach. In addition, during the course of operation, 28 cases of benign tumor were found, either during operation for such a condition or incidental to gastric surgery for other reasons. Thus there have been a total of 104 cases of benign tumor of the stomach during this period. Of the 76 cases of benign tumor of the stomach found at autopsy the classification is as follows

1	Polyps	31
	(a) multiple	16
	(b) solitary	15
2	Papillomas	8
3	Fibromas	20
	(a) simple fibromas	15
	(b) fibromyomas	5
	(c) fibroadenomas	1
	(d) fibroleiomyomas	1
	(e) fibrolipomas	1
4	Myomas	5
	(a) solitary	4
	(b) multiple	1
5	Adenomas	8
6	Leiomyomas	4

Of the 31 polyps, 5 having the gross characteristics of a benign polyp showed carcinomatous degeneration. This was found only on microscopic section. Among the adenomas there was one with carcinomatous degeneration. One leiomyoma was associated with malignancy. There was a question however as to whether it was a case of carcinomatous degeneration at the

base of the leiomyoma or a carcinoma of the stomach with an independent leiomyoma. Of the 28 benign tumors found at operation, 12 were studied microscopically. There were 6 polyps, 2 leiomyomas, 1 hemangioendothelioma, 2 fibromas and 1 fibromyoma. Of the cases found at autopsy, in only 1 instance of a polyp of the stomach was there also a solitary polyp of the colon.

Apparently, benign tumors of the stomach may be of even greater frequency than is indicated by our own figures, since many of the small benign tumors are clinically silent and therefore may escape detection during life.

An excellent description of polyps of the stomach with reproductions of their pathologic appearance is to be found in the work of Cruveilhier.¹ In Figure 2 (Plate 2) of his work there is a remarkable drawing of polyposis of the stomach. A large number of polyps of various forms and sizes may be seen. Several are situated at the cardiac end and one in the pyloric region. Those in the incipient stage are not pedunculated. In the larger ones, pedicles are present, the length of the pedicle being proportionate to the weight of the growth. The largest of the polyps are bifurcated and lobular and in some of these examination revealed the characteristics of carcinoma.

Gastric polyps may assume enormous size. In rare instances they may extend externally from the greater curvature of the stomach² and occasionally may be associated with polyps elsewhere in the intestinal tract as in several of our cases at Bellevue Hospital and as was originally de-



FIG 219 Spot film of a diverticulum of the cardiac portion of the stomach (confirmed surgically)

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Gastric polyps may assume enormous size. In rare instances they may extend externally from the greater curvature of the stomach² and occasionally may be associated with polyps elsewhere in the intestinal tract as in several of our cases at Bellevue Hospital and as was originally de-

scribed by Collier⁴ and Versé⁵ The tendency to malignant degeneration of such polyps was emphasized by Cruveilhier

A fundamental work on the subject was that of Menetrier,⁶ who divided adenomatous alterations of the gastric glands as occurring either in the form of circumscribed tumors as polyps of the stomach ("polyadénomes polypeux") or in the form of circumscribed or diffuse hypertrophic plaques ("polyadénomes en nappe"). He described the transition from irritation of the glandular epithelium through the stage of adenomatous hypertrophy and its final eventuation into carcinoma Under "polyadénomes polypeux," Menetrier referred to the discrete variety of polyp Under "polyadénomes en nappe," he included polyps arising from a single plaque and so arranged as to resemble the convolutions of the brain (Briquet) He regarded the latter rare form of polyposis as having the same pathologic background as the more common discrete, occasionally multiple, type of lesion

Other important gastric tumors are myomas, which may be pedunculated or intramural They are rounded, usually single, and originate from the muscular layer They may show considerable variation in size Fibromas may be globular or club-shaped Lipomas are much rarer, they arise from the submucosa and may project into the stomach in the manner of polyps Intragastric tumors may occur due to aberrant pancreas⁷ and cysts (Fenwick and Fenwick)

A nodule of aberrant pancreatic tissue may produce a small, rounded translucent area within the confines of the stomach having the roentgenologic appearance of a polyp^{8, 9, 10, 11, 12}

Although the aberrant pancreatic nodule in the stomach usually appears as a small, sharply defined translucent area in the pylorus, at times it may manifest itself in unusual ways It may produce an intraluminal deformity of considerable size, as it did in the pathologically confirmed case re-

ported by von Keiser¹³ in which the aberrant pancreatic mass had undergone cystic degeneration

An aberrant pancreatic mass may also be present within the wall of the stomach and produce a rigid deformity simulating malignant new growth From this standpoint Lapidari's¹⁴ observations are of considerable interest The patient, an Italian male, aged 50, gave a 3-year history of pain which occurred from 3 to 4 hours after eating, it was relieved by food In the roentgenographic examination the prepyloric antral region appeared to be slightly narrowed, irregular and rigid Extending from the greater curvature of the pyloric antrum was a fine canalicular extension well outlined by barium He concluded that the deformity was due to neoplastic infiltration, and a resection was done Operation revealed a small soft nodule, cone-shaped, about the size of a small cherry, situated in the prepyloric region toward the posterior portion of the greater curvature of the stomach The serosa covering the nodule was intact and normal in appearance Gross section of the nodule revealed on its gastric mucosal surface a small craterlike depression in the center of which was a filiform duct Histologic section revealed pancreatic exocrine tissue below the gastric mucosa with the small filiform tract in its center, opening into the stomach and in turn draining small excretory ducts running into the pancreatic tissue There was an associated hypertrophy of the tunica muscularis of the stomach The duct was surrounded by Brunner's glands The tissue was normal in appearance and definitely not neoplastic in nature It was the small filiform duct which when filled with barium had given the appearance of a fine canaliculus in the roentgenogram

Lapidari in this thoroughly documented study not only showed that an aberrant pancreas may produce a deformity of the pylorus simulating that of malignant invasion but he also called attention to the roentgen demonstration of the filiform duct, the potential diagnostic significance of

which was understood only on the basis of pathologic study of the resected lesion

A crater in an aberrant pancreatic tumor of the stomach may be in the form of an umbilication without actual ulceration, and the entire tumor, including the central depression, may be covered by normal gastric mucosa. This characteristic had been observed by a number of authors but was particularly emphasized by Benner¹. In the five cases which he described this central crater appearing like a pseudodiverticulum had a depth which varied from 0.5 cm. to 1 cm. to about the level of the boundary between the mucosa and the submucosa. In the submucosal connective tissue beneath the pouches was well differentiated pancreatic tissue with ductlike structures. The duct arrangement was haphazard, but in some cases the ducts appeared to join the gastric mucosal glands in the depths of the pseudodiverticula. As Benner pointed out, the formation of the crater may be the result of traction produced by the pancreatic nodule upon the overlying mucosa.

Therefore an intramural nodule with a pseudodiverticulum might be strongly suggestive anatomically of aberrant pancreas. However, as valuable as this characteristic might be in the examination of the gross lesion if barium were trapped in this umbilication, appearing as a niche in the roentgenogram it would not be helpful in differentiating the tumor from other new growths which tend to form craters. Suggestive roentgen evidence which might aid in the diagnosis of a pancreatic tumor would be the demonstration of a filiform ductlike structure within the mass which communicated with the stomach as in the case described by Lapidari. Umbilication in a pancreatic nodule in the stomach is well illustrated by the gross appearance and the microscopic section in the case reported by Joyeux, Guibert and Guenier¹⁶ as well as in the contribution by Littner and Kirsh^{16A}. An aberrant pancreatic nodule may be a cause of pyloric obstruction and ulceration.¹

Carcinoid tumors of the stomach may

show only as an intraluminal area of translucency roentgenologically indistinguishable from a polypoid lesion benign or malignant. In the case described by Wirts and Breckenbridge¹⁸ the gastric deformity simulated that of a malignant adenoma. The diagnosis of carcinoid was based entirely on histologic study. Actually, the malignant nature of the lesion was indicated by the fact that metastases were found in 5 of 11 sections of lymph nodes.

The nature of the carcinoid tumor will be described more fully later in the discussion of tumors of the duodenum.

An anatomic feature of importance radiologically and clinically is the fact that benign tumors of the stomach not infrequently show evidence of ulceration which may lead to a slow but persistent seepage of blood or actual massive hemorrhage.

ROENTGEN DIAGNOSIS

The diagnosis of tumors of this nature depends upon their pathologic characteristics and therefore will give the following evidence of their presence in the roentgenogram.

1. Benign tumors are usually of rounded contour. Therefore their displacement of the barium filled stomach will give rise to a well rounded circumscribed translucent area. Since such tumors are intraluminal in location their presence may readily be obscured when too much of the barium suspension is introduced into the stomach. The thin layer method of examination is therefore of great importance particularly when the lesions are small in size. Occasionally the tumor has a lobulated surface and a corresponding degree of irregularity of contour of the translucent area will be produced in the roentgenogram. When the lesion is multiple in character in cases of diffuse polyposis, there will be correspondingly small fairly well rounded, circumscribed translucent areas throughout the zone of gastric involvement. Flecks of barium may adhere to the interstices between the lobu-

lated portions or the discrete polypoid areas of the tumor. The first example of the mottling in the roentgenogram produced in a case of gastric polyposis was published by Meyer.¹⁹

Roentgen examination in his case revealed an almost complete obliteration of the pars pylorica and the pars media of the stomach. The mottling in the involved region suggested numerous irregular masses. The specimen at autopsy showed the entire mucosa, except for a small area at the cardia, to be covered with polyps. Near the pylorus was a large, grapelike bunch of polyps invaginating the pylorus. Throughout the stomach there was marked chronic gastritis.

An excellent example of the "polyadénomes en nappe" type of gastric polyposis is to be found in the report by Balfour.²⁰ Roentgenologically, there were numerous small translucent areas in the distal half of the stomach, produced by the displacement of the barium by the many small polyps demonstrable in the resected specimen. The pathologist considered the lesion as non-malignant.

2. Benign tumors, as a rule, do not invade the wall of the stomach. This, therefore, leads to another important roentgen characteristic, that is, an absence of any evidence of irregularity in the contour of the stomach and the persistence of peristaltic activity through the region of origin of the tumor. In rare cases, however, there may be an absence of gastric peristalsis in the presence of diffuse polyposis.²¹ Because of the fact that the wall of the stomach is not ordinarily involved, particularly in the case of the solitary tumor, the mucosal folds remain intact. This obviously does not hold in the case of a diffuse polyposis, since in these cases the numerous translucent areas are actually due to hypertrophic adenomatous proliferations of wide areas of the gastric mucosa itself.

3. Because of the fact that benign tumors are frequently attached by a pedicle, they exhibit a considerable degree of mobility. Therefore, under fluoroscopic control it may

be possible to move the tumor about with changes in the position of the translucent zone corresponding to it.

4. When the mobility of the tumor is considerable, because of the presence of a long pedicle, it may actually intussuscept through the pyloric ring into the duodenum. On fluoroscopic and roentgen examination, therefore, the translucent area produced by the tumor may be found within the duodenum itself, even though it is primarily gastric in origin. A knowledge of the fact that such mobility is a characteristic of benign tumors will put the examiner on his guard, and therefore he will not be misled into assuming that he is dealing with a duodenal tumor of intrinsic origin. Moreover, in some cases, it may be possible manually to displace the tumor back into the stomach under fluoroscopic observation. Roentgen examination will also throw considerable light on the mobility of the tumor, and in a series of films it may be found to vary its position considerably. Thus at one time the translucent area may be present in the duodenum. At another time the duodenal bulb may be normal, but the tumor may be shown to have shifted into the gastric area. Thus, there will be considerable variation in the appearance and the actual location of the intragastric translucency, depending on the shifting changes in the position of the tumor itself.

In Rigler's²² interesting case of a benign papilloma of the antrum of the stomach, roentgen study clearly demonstrated the free mobility of the tumor. In the original roentgenogram, the translucent area produced by the tumor in the barium-filled stomach was noted in the pyloric antrum. After manipulation of the tumor, the roentgen defect was then seen in the immediate prepyloric region. Finally, the tumor was forced through the pyloric ring into the duodenum, and roentgen examination at this time showed a central, fairly well-rounded translucent area within the duodenal bulb. Operation confirmed the diagnosis of a tumor which, although attached to the an-

trum, could be easily pushed through the pyloric ring into the duodenum

5 When the pull on the stomach by the tumor is extreme there may be an invagination of the gastric wall into the lumen of the stomach. In rare cases, the gastric mucosa may actually become intussuscepted through the duodenum.^{3, 24} When the gastric wall invades the lumen of the stomach there will be an irregularly outlined translucent area corresponding to this region in addition to the defect produced by the tumor itself.

6 In rare instances the pedicle of the tumor may be demonstrable roentgenologically, as exemplified in *Natorps case*. The examination was made by a combination of barium administration and air injection. Not only was the translucent rounded area produced by the polyp visible in the roentgenogram of the stomach but, in addition, the pedicle from which the polyp hung could be clearly seen. The roentgen findings corresponded exactly with the appearance of the resected specimen including the pedicle.

7 Another important feature in the pathologic appearance of a benign tumor of the stomach is the presence of an ulcer, frequently at or near its apex. When barium is lodged within this ulcer, it may be recognizable as a niche in the roentgenogram. Therefore one may be able to determine not only the boundaries of the circumscribed benign tumor itself, but, in addition a niche may be seen in direct relation to it. This feature is not only roentgenologically significant but in the presence of bleeding will indicate its probable origin. The niche may be stellate in appearance corresponding to the irregular character of the ulceration itself.

The presence of a niche due to ulceration within a benign tumor is beautifully shown in the case of a gastric neurinoma described by Odquist.⁶ In one of Renander's cases roentgen examination showed a large filling defect within the pars pylorica constant on repeated examination during the course of a year. A small fleck was noted within this

translucent zone having the appearance of barium in a crater. Operation revealed a submucous lipoma with a crater from which the bleeding had taken place. In Senturia's²⁵ excellently documented case of a gastric neurinoma the roentgenogram showed a well defined, rounded, intraluminal translucent area with a large 'en face' niche. A picture of the tumor showing the crater and a photomicrograph are included.

Although ulceration with the production of a niche in the roentgenogram may occur in the presence of a leiomyoma, this finding is therefore by no means limited to such tumors. The niche has also been noted as part of the roentgenographic deformity of a schwannoma.⁷ In one of the schwannomas of the stomach reported by Ludin³⁰ there were several deep seated ulcerations. Although the roentgenogram in this particular case did not show any evidence of a niche within the defect produced by the tumor it is conceivable that under more propitious circumstances one or more of the craters might have retained enough barium to have become recognizable roentgenologically. It has also been found in a paraganglioma³¹ in a lipoma^{3, 33} and in a neurofibroma.³⁴ Ulceration may also occur in hemangioma of the stomach.⁷ Probably the only reason a niche within a benign tumor is more apt to represent an ulcer within a leiomyoma is because of the comparative frequency of this type of benign submucosal tumor. Therefore the niche represents non-specific evidence of necrosis within a benign tumor.

8 In rare instances areas of calcification have been found within a leiomyoma of the stomach. Heitz⁷ described such findings in a leiomyoma found incidentally at autopsy in a patient who died of bronchopneumonia. The tumor felt very hard. Areas of calcification were found throughout the muscular tissue. He stated that Cornil and Ranvier had reported finding calcification in a few cases of gastric leiomyoma.

In a study of 50 autopsies chosen at random Meisner³⁷ found one or more leiomyomas in 23 of the 50 stomachs which he

within the pylorus and in some cases actually invaginate through the pylorus into the duodenum ⁴⁰

Considerable impetus to the investigation of the subject of prolapse of pyloric mucosa into the duodenum resulted from a number of publications. Pathologic evidence supporting the contention that folds of gastric mucosa may prolapse into the duodenum was furnished by Manning and Gunter ⁹. They included cross sections of the pyloro-duodenal segment obtained at autopsy in 6 cases. However, they stated that a certain looseness of the mucosa in the antral region is normally present but that in some cases the redundancy of the folds is such that they may be pulled through the pyloric canal into the duodenum.

It is my considered opinion that the condition is far less frequent than one would be led to expect from the increasingly large number of publications on the subject. Many of the roentgen findings relied upon to substantiate this diagnosis may be considered as variations of normal structure and function.

1 The 'umbrella or mushroom effect' that is referred to frequently in support of the diagnosis may actually represent a cross section of the base of the bulb. The elliptically outlined translucent area actually represents the lumen of the bulb itself as visualized by direct inspection of the base. Radiating folds of normal mucosa at the base of the bulb may then produce the umbrella appearance.

2 The appearance of gastric folds within the confines of the proximal portion of the duodenum may actually be due to foreshortening in the roentgenogram. It must be kept in mind that the three dimensional pyloroduodenal segment has been transfixed on a two dimensional film. The relation of the pylorus to the base of the bulb will then depend (1) on the anatomic position of this area in relation to the direction of the radiographic exposure. Thus in the postero-anterior position the base of the bulb may appear to overlap partially the pyloric

antrum so that the intervening pyloric ring is obscured. In the right oblique position the configuration of the pyloroduodenal area may be brought more clearly into view. Because of the manner in which the duodenum may overlie the pylorus strands of gastric mucosa may appear in the two dimensional roentgenogram as if they were actually present within the confines of the bulb itself. (2) Another factor in the relation of the pylorus to the duodenal bulb is purely anatomic, depending on the varying directions of the bulb within normal limits. It may be directed laterally, medially, posteriorly or anteriorly. The mere position of the bulb itself, independently of the direction of the x rays, may in this manner cause partial overlapping of the pylorus by the duodenal bulb, with consequent simulation of an intraduodenal protrusion of gastric mucosa.

3 The concave defect at the base of the bulb to either side of the pyloric ring is another sign which is referred to sometimes in order to substantiate the diagnosis of prolapse of pyloric mucosa. However, these findings may be simply the result of the propulsive effect of the pylorus initiated by vigorous peristalsis against the base of the bulb. This pyloric thrust is entirely a functional phenomenon. The appearance may be compared with the invagination of a normal cervix. Such evidence in the roentgenogram cannot be interpreted justifiably as an indication of actual prolapse of pyloric mucosa. It is reminiscent rather of the findings in hypertrophy of the pyloric muscle and in hypertrophic pyloric stenosis in which there is no suspicion of prolapse of pyloric mucosa. As a matter of fact the occlusion of the pyloric outlet in these cases would mitigate against such a prolapse taking place.

4 Moreover, even if the forceful propulsion of the pylorus produces an area of transient translucency near the base of the bulb it may be considered a purely functional phenomenon and the significance of the findings must not be exaggerated as

constituting evidence of a clinically significant pathologic entity

5 Translucencies within the duodenal bulb may be due to irregular and incomplete filling with barium. It is not necessary to assume that they are due to the intraluminal invasion of gastric mucosa.

6 Pressure of the duodenal bulb against the spine or other neighboring structures may produce translucencies.

7 Rounded intraluminal translucencies of a discrete character persistently present within the duodenum may be due to the duodenal mucosa itself, either during periods of transition in the changing pattern of a normal mucosa, or of a permanent character as a result of inflammatory changes due to an actual duodenitis. Normal duodenal folds caught in cross section during roentgenologic examination may appear as rounded translucencies.

8 Some of the support of the diagnosis is based on surgical findings not always of a conclusive nature. At the time of operation it may be possible manually to invaginate partially the prepyloric portion of the stomach into the lumen of the duodenum, particularly if accompanied by a cufflike overriding of the pylorus by the bulb. There is no proof that such evidence indicates that during life there is a pathologic propulsion of pyloric mucosa into the duodenum. Also, when the pyloric segment has been cut through as a result of a subtotal gastrectomy the mucosa just proximal to the cut

edge may be pulled up quite easily by means of forceps and may exhibit a considerable range of back-and-forth mobility. However, this may be due entirely to the operative procedure itself, so that the pylorus is no longer firmly fixed to the more distal mucosa from which it has been severed.

Most of the roentgen evidence that I have seen which purports to substantiate the diagnosis of prolapse of gastric mucosa into the duodenum can be reasonably explained on the basis of the normal anatomy and functional behavior of the pyloro-duodenal segments.

Probably the disorder is quite rare. Certainly the diagnosis should be made with considerable circumspection. Only in the event that the apparent abnormality cannot be explained rationally on the basis of normal phenomena should the diagnosis be entertained of prolapse of gastric mucosa into the duodenum. This approach is particularly important, since operative intervention has been recommended as a therapeutic procedure in some of these cases.

Illustrative Cases of Benign Tumors

M. D., female, aged 65. During the preceding 4 or 5 weeks the patient had complained of a dull, constant, nonradiating epigastric pain lasting from a few minutes to an hour. There was no vomiting and no blood in the stools. The patient lost 15 pounds in the preceding 3 months. She had had a cholecystectomy 14 months previously and had been well until this recent episode. Physical examination was essentially negative except for a large ven-

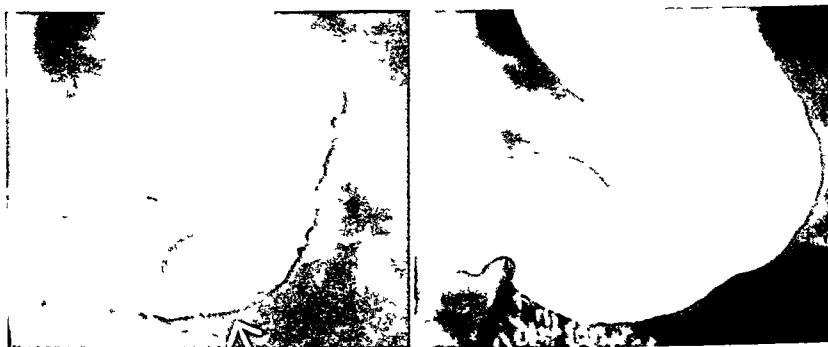


FIG. 220 (A, *Left*) Benign tumor of the stomach. (B, *Right*) Same patient. The tumor is not visible with the stomach distended with barium.

tral hernia. Operation revealed a benign tumor on the greater curvature of the stomach about the size of a golf ball. A local removal of the tumor was done.

Pathologic examination revealed an encapsulated ovoid mass $4 \times 3 \times 1$ cm. The diagnosis was leiomyoma of the stomach.

Roentgen examination (Fig 220 A) showed evidence of a benign tumor based on the following characteristics: (1) the translucent area was intraluminal, (2) it was well rounded, (3) there was no interruption of gastric peristalsis. When the stomach was completely distended with barium, the tumor could not be seen (Fig 220 B).

In the next case not only was the polyp visible in the roentgenogram, but also, apparently, the pedicle itself.

M. H., female, aged 65. The patient complained of dysphagia, moderate weight loss, occasional blood in the stool, pallor, dizziness and weakness during the preceding year. She had no abdominal pain. Physical examination revealed an elderly female with extremely marked pallor. The thyroid gland was enlarged (apparently the cause of the dysphagia). Abdominal examination was negative. The hemoglobin was 30 per cent, the R. B. C., 3,000,000.

Gastric analysis showed an absence of free hydrochloric acid.

At operation a polyp was found at the greater curvature of the stomach in the distal portion of the pars pylorica. The polyp measured approximately 2 cm \times 1 cm and was attached to a small pedicle about $\frac{3}{4}$ cm long (Fig 221 A).

The report of the macroscopic examination was as follows: Specimen consists of a

pedunculated piece of tissue 2×1 cm which is oval, hanging from a stem attached to a base which is $2 \times 2 \times 1$ cm. On section, the base is white and the tumor part is reddish brown.

The report of the microscopic examination was as follows: 'The base consists of mucosa and submucosa. The mucosa is typical gastric mucosa. A fibrous tissue stem extends out to the tumor. This stem contains blood vessels and occasional dilated acini lined with mucous cells. The mass itself consists of dilated acini lined with mucous cells. There is considerable inflammatory cell invasion of the interacini tissues and edema is present.'

The diagnosis was benign gastric polyp.

Roentgen examination revealed a small, rounded, well circumscribed translucent area within the confines of the pars pylorica having the roentgen characteristics of a benign tumor (Fig 221 B). In addition to the rounded, more centrally placed translucent area, there was another translucent zone attached to the greater curvature.

In viewing the appearance of the pathologic specimen, it is possible that this latter area of translucency was due to the base to which the pedicle of the polyp was attached.

The importance of the thin layer technique in making a small polyp visible is shown in the following case.

C. F. female, aged 50 (Fig 222). Note the small sharply defined rounded translucent area in the pars media produced by the polyp. This was almost completely obscured when the stomach was distended with barium. The diagnosis on pathologic examination of the resected tumor was polyp of the stomach.

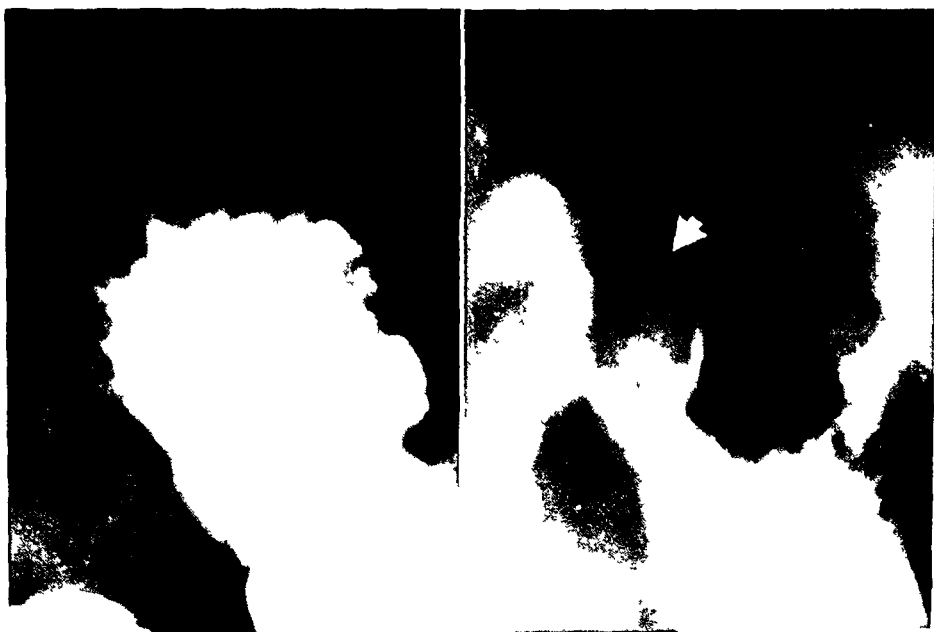


FIG 221 (A Left) Appearance of the resected polyp (B Right) Same patient. The translucent areas are produced by the polyp.



FIG 222 (*Top*) Small polyp of the pars media of the stomach seen with thin layer technic. Note the small sharply defined intraluminal area of translucency corresponding to the polyp which was found at operation.

FIG 223 (*Bottom*) Leiomyoma of the pars cardiaca of the stomach. (A, *Left*) Examination in the prone position. The tumor is not visualized because of overdistention of this area with barium. (B, *Right*) Examination in the erect position so that the gastric air bubble is not obscured. Note the sharply defined, rounded soft-tissue shadow of the pars cardiaca hugging the lesser curvature, produced by the leiomyoma which was resected at operation.



Even fairly large benign tumors of the stomach may be obscured because of overdistention of the involved area with barium. This fact is illustrated not only by Figures 220 A and B but also by the leiomyoma of the pars cardia of the stomach in the following case.

K J, female, aged 60. The patient gave a history of upper abdominal discomfort, apparently of 5 months' duration. At the onset she had burning epigastric pain and vomited

some black material. This was followed by three watery black stools. Her family physician told her that she might have an ulcer and treated her on that basis. The pain was made worse by hunger and was alleviated by eating and antacids.

Physical examination was essentially negative. The diagnosis was based on the roentgenographic evidence. Figure 223 A shows the appearance of the pars cardiaca when filled with barium. There is no evidence of tumefaction. Figure 223 B, in the erect position so that the gastric air bubble is not obscured,

shows evidence of a well defined, rounded soft tissue mass hugging the lesser curvature of the pars cardiaca. The preoperative diagnosis was carcinoma of the cardia of the stomach. A thoraco abdominal exploration revealed an irregular lobulated intramural tumor on the anterior surface of the cardia of the stomach. The tumor was projected into the lumen in a polypoid manner. There were no mediastatic nodes in the celiac region. A longitudinal incision was made in the stomach wall and the tumor was palpated from within. A section of the wall of the stomach involving the tumor mass was removed. Study of a frozen section was reported as showing no evidence of malignancy. A local excision of the tumor was performed.

Macroscopic examination "Specimen received fixed consists of a small well demarcated mass 2.5 x 1 cm found beneath the mucosa. It does not appear to invade the surrounding tissue. Upon section the color is white, the tissue firm.

Microscopic examination Normal gastric mucosa and segment of squamous cell mucosa of the esophagus is seen. Lying in the submucosa and internal to the outer layers of normal muscle there is a circumscribed mass of tissue which consists of an irregular pattern of interlacing strands and whorls of spindle cells. Trichrome stain reveals these to be of muscle origin.

Diagnosis *Leiomyoma of cardia of stomach*

In this case therefore the diagnosis of a tumor depended on the fact that in the examination in the erect position it was possible to visualize the soft tissue shadow produced by the mass partially encompassed by a rim of barium and standing out clearly against the contrast of the gastric air bubble. However when the air bubble was displaced by the barium which distended the pars cardiaca in the prone position the deformity caused by the tumor was obscured.

Of unusual interest in connection with the subject of gastric polyps as part of a diffuse polypoid involvement of the digestive tract are the observations of Jehgers, McKusick and Katz.⁵¹ They collected 10 cases including 2 of their own which exhibited a peculiar syndrome. In addition to generalized gastrointestinal polyposis these patients showed a distribution of melanin spots present mainly on the

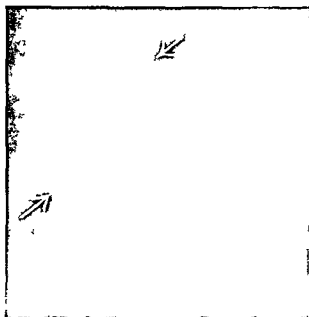
lips, the oral mucous membrane and the digits. Moreover, there was a significant clinical background suggesting a hereditary predisposition to these outstanding features of this condition. Intussusception and hemorrhage occasionally massive, resulted from the intestinal polyps. In some cases the polyp became malignant.

Wolff's case⁵ reported in 1952 is very interesting.

The patient was a male aged 50 who gave a history of episodes of abdominal pain. Between the ages of 17 and 23 he was operated on four times for polyps of the small intestine associated with intussusception. Several polyps were removed with resection of a loop of small intestine. Two years later there was a recurrent intussusception again necessitating resection of a portion of the small intestine containing many polyps. Finally at the age of 50 he underwent another operation. One polyp was removed from the duodenum. Another polyp was removed from the sigmoid colon. A portion of jejunum was also removed. It contained several sessile and pedunculated polyps. In addition there were other polyps in the small intestine and the large intestine which were not removed. Physical examination revealed marked pigmentation of the upper lip which had been present since childhood. It was significant that a son died when 3 years old following an operation for intestinal intussusception. He had had dark spots on the lips and the face since birth. A 5 year old daughter had pigmentation of the face and the forehead especially about the nose, the lips and the mouth and also on the inner aspect of the cheeks, the thumbs, the fingers and the forearms. A son aged 16 showed similar pigmentation.

The first definite reference in the literature to the association of gastrointestinal polyposis and pigmentation was by Peutz of The Hague in 1921. He described a family in which seven members during a period of three generations exhibited this strange syndrome of intestinal polyposis and pigmented spots distributed over the mouth, the hands and the feet. Observations of a similar nature with a familial predisposition were published by Dijk and Oudendal, Foster and others. These pigmented spots appear as round, oval or irregular patches. In color

FIG 225 (A, *Top*) Gastric polyp intussuscepting into the duodenum (B, *Bottom, left*) Same patient Ulcerating area on the fundus of the tumor (C *Bottom right*) Same patient Pedicle of the tumor, 3 cm wide



mors in that segment The involved portion of the jejunum was resected The excised loop of jejunum contained a total of 5 polyps Figure 224 C shows the gastric polyp and one of the jejunal polyps

Pathologic diagnosis (1) Adenomatous polyp of the stomach (2) Adenomatous polyps of the small intestine

This then is a case of a polyp of the stomach and multiple polyps of the jejunum in a boy exhibiting melanin pigmentation mainly perioral in distribution Although the exact relationship of these features is not well understood the clinical significance of the pigmentation in calling attention to the possible existence of associated gastro intestinal polyposis was emphasized by the pathologic findings in the patient

In some cases the gastric polyp may intussuscept through the pyloric ring and be found within the duodenal bulb Under such

conditions, the lesion although originating in the stomach may simulate that of a primary intraduodenal tumor This is illustrated by the following case

T G aged 51 The patient entered the hospital complaining primarily of a discharge from the rectum This was found on examination to be due to a fistula in ano In addition, he presented a picture of marked secondary anemia the red blood cells varied from 2 200 000 to 2 600 000 on five examinations The hemoglobin was 30 per cent No stippling or nucleated red blood cells were present Although he had absolutely no abdominal symptoms or palpable masses he was roentgenographed in an effort to determine the cause

At operation on delivering the pylorus a mass the size of an orange was found in the stomach This mass was freely movable and attached to the stomach wall near the greater curvature There was no evidence of a mass

in the duodenum. The stomach wall was opened, and a polyp was delivered, which was about 1.5 cm in diameter. There was an ulcer over the fundus, which had eroded through and was causing bleeding. The attachment of the polyp was about 2½ inches from the pylorus on the greater curvature. The pedicle was long enough to allow the polyp to enter the duodenum.

Roentgen examination (Fig 225 A) showed the large, rounded translucent area to be within the duodenal bulb, the barium surrounding its outermost limits. A diagnosis of polyp within the duodenum was made. At operation, however, the tumor was found to be within the stomach, attached by a long pedicle. Presumably, then, the tumor which had intussuscepted through the pylorus into the duodenum had, prior to abdominal exploration, slipped back into the stomach. The appearance of the polyp is shown in Figure 225 B and C. Note the ulceration in the polyp from which bleeding had occurred.

The following is another interesting example of a gastric polyp which had intussuscepted into the duodenum.

E. H., aged 59. This patient had been complaining of abdominal pain for about 14 months. He had lost 30 pounds in the preceding 3 months. The pains were colicky, in the epigastric region, and had radiated to the back and along the right costal border. The pain was not related to food and was not relieved by soda. There was no vomiting. He had noticed tarry stools during the preceding 1½ months.

During the physical examination inspection and palpation revealed a definite mass in the epigastrium slightly to the left of the midline. The mass was about 4 or 5 cm in diameter, firm to the touch and moderately tender. The liver and the spleen were palpable.

At operation, in the lumen of the first portion of the duodenum was a 5-cm nodular, hard mass. The lymph nodes along the mesenteric curvature were extensively infiltrated, with invasion along the posterior gastric wall. There were milium malignant nodules over the anterior gastric wall involving the serosa. In the gastrohepatic omentum there was a malignant mass measuring 3 cm. It could not be definitely determined whether the involvement of the posterior gastric wall was or was not an intrinsic gastric lesion. The opinion on palpation was that the mass was extragastric. The pyloric antrum was defi-

nately not involved by malignant tissue, making certain that there was no direct extension into the duodenum from a gastric lesion. The extensiveness of involvement of the gastric wall and the rarity of duodenal carcinoma, however, made it possible that this was a primary gastric carcinoma. Resection was found to be impossible. A fragment of malignant tissue was taken from the greater curvature of the stomach. Microscopic examination revealed evidence of carcinoma.

The autopsy report was as follows: "On the posterior wall of the stomach, about midway between the cardia and the pyloric ring, is a large, irregular, hard tumor mass overlying the Magenstrasse. The mass projects high above the surface of the mucosa and infiltrates over the posterior wall from the cardia to the pyloric antrum and includes the lesser curvature. The mass is very friable. Suspended on a freely movable stalk, attached to the prepyloric wall, is a large, fungating, polypoid, irregular mass, which is prolapsed through the pyloric ring and partially obstructs the lumen of the duodenum. The polypus is soft, and small areas of ulceration may be seen on its surface. The duodenum and the small and large bowel show no pathology other than some post-mortem discoloration. The periaortic and retroperitoneal nodes are somewhat enlarged and firm. Metastatic invasion of the upper abdominal nodes is seen. There is, in addition, metastatic carcinoma of the liver, adrenals and renal pelves."

The report of the microscopic examination was: "The normal mucosa has been entirely replaced by masses of fibrous stroma throughout which strands of epithelial cells infiltrate. The muscular coat has also been invaded. Small foci of necrosis are present in the mucosa. The polyp stalk itself shows no evidence of tumor infiltration. Sections through the wall of the stomach between the tumor mass and the polyp show infiltrating carcinoma extending to the base of the polyp."

The diagnosis was primary undifferentiated carcinoma of the stomach with a benign pyloric polyp (Fig 226 A).

Roentgen examination (Fig 226 B) revealed the presence of a large, translucent, irregularly outlined area within the duodenal bulb characteristic of benign tumor. The possibility was considered that this represented a gastric polyp which, because of its long pedicle, had intussuscepted through the pyloric ring into the duodenal bulb. The whole appearance was that of a fungating tumor.

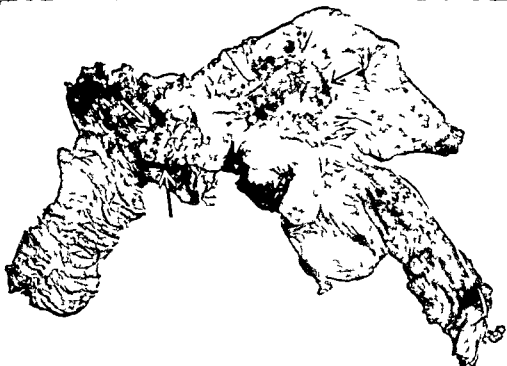


FIG 226 A Prepyloric polyp intussuscepting into the duodenum indicated by the 2 arrows to the left In addition there is a malignant lesion on the posterior wall of the stomach indicated by the single arrow to the right

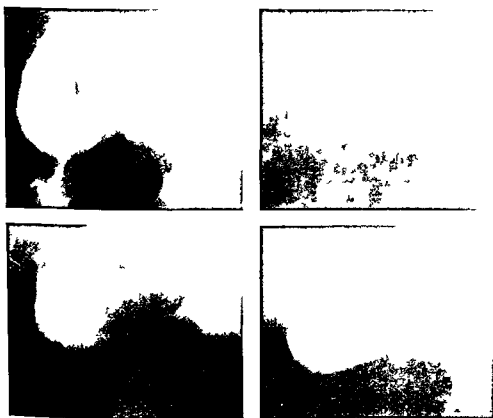


FIG 226 B Same patient as is shown in Figure 226 A Note the deformity produced by the polyp which had intussuscepted into the duodenum

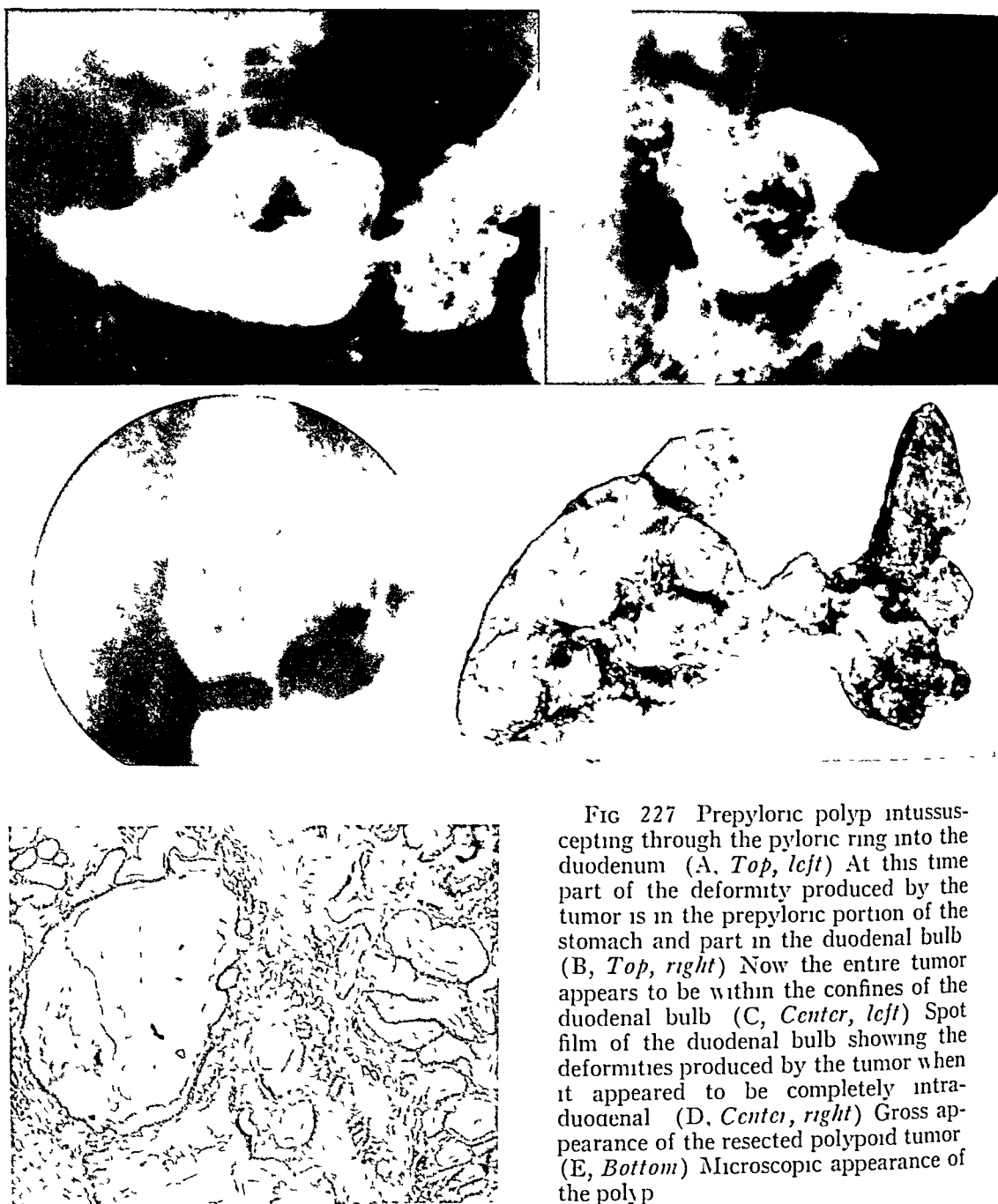


FIG 227 Prepyloric polyp intussuscepting through the pyloric ring into the duodenum (A, *Top, left*) At this time part of the deformity produced by the tumor is in the prepyloric portion of the stomach and part in the duodenal bulb (B, *Top, right*) Now the entire tumor appears to be within the confines of the duodenal bulb (C, *Center, left*) Spot film of the duodenal bulb showing the deformities produced by the tumor when it appeared to be completely intra-duodenal (D, *Center, right*) Gross appearance of the resected polypoid tumor (E, *Bottom*) Microscopic appearance of the polyp

with polypoid excrescences. No diagnosis was made of the malignancy of the stomach which was found at operation. Because of the fact that the tumor within the duodenum was considered sufficient explanation for the physical findings, no attempt was made to study the stomach with greater detail in view of the fact that, grossly, there was no definite evidence of organic abnormality.

The reason why the intraduodenal tumor

was believed to have originated from the pyloric side and intussuscepted into the duodenum was the rarity of primary tumors that originate entirely within the duodenal bulb. Note how closely the roentgen findings within the duodenal bulb correspond to the anatomic appearance of the polyp itself.

Sometimes the actual process of intussusception of a prepyloric tumor into the duo-

denum may be noted in the radiographic examination as illustrated in this case

M B, female, aged 52 This patient gave a 1 year history of recurring attacks of severe knifelike pain in the left upper quadrant lasting from one half hour to one hour One particularly severe attack kept her awake during the night The pain was not related to food intake There was a moderate weight loss

Nine years previously she had a hemorrhoidectomy She continued to notice blood in her stools from time to time There were two essential findings on physical examination (1) a palpable mass in the left upper quadrant (2) many large hemorrhoids

Roentgenographic examination revealed evidence of a polypoid tumor which at one time was partly in the pylorus and partly in the duodenum In Figure 227 A note the irregularly outlined translucent area in the prepyloric portion of the stomach as well as intraluminal translucent areas within the confines of the duodenal bulb At other times evidence of the tumor was recognizable only within the duodenum (Fig 227 B) Figure 227 C represents a spot film of the pyloroduodenal area Note the translucent areas within the confines of the duodenal bulb On the basis of these findings a preoperative diagnosis of a prepyloric tumor with intussusception into the duodenal bulb was made The gross nature of the tumor, as well as its anatomic region of origin were confirmed by surgical exploration and local removal through a gastrotomy opening

Pathologic examination The specimen consists of an irregular polypoid mass measuring 7 cm in length and 4 cm in its largest width The mass is pedunculated and adherent to a small irregular piece of congested gastric mucosa The pedicle measures 1 cm in length and 0.5 cm in width The polyp itself has a lobular architecture and is pink in color It is covered by a hyperplastic slimy mucosa and shows focal areas of hemorrhage and ulceration on the surface On section the polyp is grayish white in color

Microscopic diagnosis Edematous mucous gland adenomatous polyp

Figure 227 D shows the gross appearance of the tumor Figure 227 F is the microscopic appearance

In this case therefore it was possible to demonstrate radiographically the actual migration of the prepyloric polyp into the duodenum

In some cases, the extreme mobility of a

benign gastric tumor may be shown by its remarkable changeability in location In addition, the ulceration occasionally found in such tumors is well illustrated by the following case

E E, female, aged 58 About 4 days before admission to the hospital this patient began to have fainting spells and vomited on an average of once a day for a few days Physical examination of the abdomen was essentially negative

Operation disclosed a firm, round mass, the size of a golf ball, covered with mucosa and lying freely in the lumen of the stomach and attached by a pedicle to the cardiac end, at the greater curvature on the posterior wall This mass could be easily pushed to the pyloric region the cardiac end of the stomach pushing inward In the mucosa just along the side of the pedicle of the polyp was an ulcerated, sharply defined area about $\frac{1}{2}$ inch in diameter The polyp was removed

The report of the macroscopic examination was as follows "The specimen of tissue from the stomach consists of a spherical, circumscribed tumor beneath an oblong piece of mucosa, the outline of which measures 6.5 cm in length and 4.3 cm in width The tumor itself measures 4.2 cm in diameter, is soft, not readily movable, and is covered by a bluish pink flattened pebblelike mucosa At its summit, and also toward the flap of loose mucosa there are two deep ulcerations having the diameters of 2 cm and 4 cm respectively About these ulcerations the mucosa is raised and irregular Inferior to the summit ulceration there are three yellowish, firm raised areas measuring 8 mm in diameter On the loose portion of mucosa there is a large, deep ulceration having a smooth, flat yellowish base which measures 2 cm long 0.6 cm in width and 0.5 cm in depth The margin is raised and is rounded into the ulceration No point of hemorrhage or bleeding is discernible Also in the mucosa toward the tumor are two smaller depressions which do not penetrate the mucosa These measure 1 x 0.3 x 0.2 cm and 0.5 x 3 x 0.2 cm On cut section the tumor is a circumscribed encapsulated spherical mass measuring 3.8 cm in diameter It is separated from the overlying mucosa by loose connective tissue At no point does the tumor show invasion through the capsule The substance of the tumor consists of whirling bundles of red tissue throughout which there is an admixture of pearly white strands Its vascularity is indicated by a diffuse, fine, red stippling

throughout. The small, raised, yellowish nodules already described occupy the entire thickness of the mucosa but do not appear to be contiguous with the underlying tumor mass."

The microscopic examination revealed the tumor to be a leiomyoma of the stomach.

Roentgen examination in the erect position (Fig 228 A) revealed a rounded translucent area, apparently occupying almost the

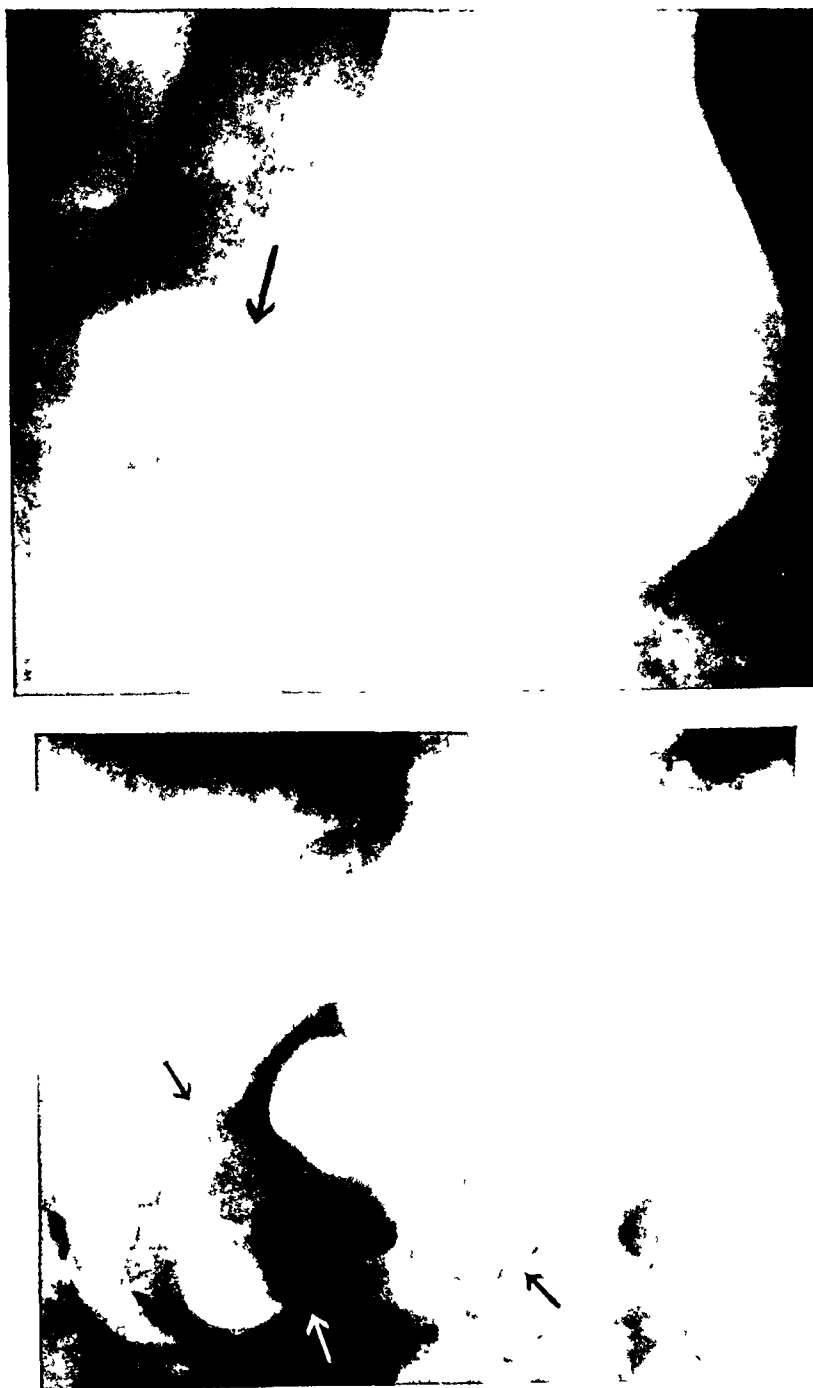


FIG 228 (A, *Top*) Gastric tumor within the duodenal bulb (B, *Bottom*) Same patient. The tumor is now present along the greater curvature of the stomach.



FIG 228 (C, *Left*) Same patient. At this time the tumor occupies the pyloric antrum. Note the fleck of barium in its center, apparently representing the niche in an ulcer of the tumor. The translucent area in the pars media probably represents partial gastric intussusception. (D, *Right*) The leiomyoma of the stomach removed at operation. The arrow points to an ulcer in the tumor.

entire lumen of the duodenum. The translucent area was about the size of a golf ball. It was circumscribed by a layer of barium of variable thickness. There was an area of narrowing proximal to this region. Examination in the prone position (Fig 228 B) revealed considerable change in the exact relationship of the translucent area to the rest of the stomach, indicating considerable mobility of the tumor mass. At this time the tumor was situated at the greater curvature side of the stomach in its distal area. A film taken in the prone first oblique position (Fig 228 C) showed, in addition to the translucent area occupying almost the entire pylorus, a central dense fleck of barium and another similar fleck smaller in size near the periphery, probably the result of the presence of barium within ulcerations in the tumor. There was in addition a translucent area in the pars media, apparently due to invaginated gastric mucosa resulting from the pull produced by the pedicle to which the tumor was attached. The change in location of the translucent area in the various observations was due to the mobility of the tumor, which was present in the stomach at one time and in the duodenum at another. The appearance of the tumor itself is noted in Figure 228 D. Note the crater near the apex of the leiomyoma.

The clinical significance of an ulcer in a benign tumor is illustrated by the next case.

A K, male, aged 50. The patient had been well until 2 days before his admission to the hospital when he developed crampy epigastric pain which finally became very severe. The following day he vomited dark blood and had 4 or 5 loose, black stools, followed by collapse. There was no weight loss or any previous history of tarry stools. Physical examination revealed a pallid middle-aged white male. Abdominal examination was essentially negative.

At operation, a large, smooth, sessile, moderately movable mass was found in the prepyloric region. There were no palpable regional lymph nodes or any evidences of metastases. A partial gastrectomy was done.

Gross examination of the specimen (Fig 229 A) was reported on as follows: On opening the specimen an oval tumor mass measuring 5 cm x 4 cm is found on the anterior wall near the lesser curvature. The tumor has an elastic consistency and is covered by smooth mucosa. There are two ulcers measuring 3 cm in diameter in the overlying mucosa. The normal rugal pattern is lost and the whole mucosa is smooth. On bisecting the tumor it is found that the tumor is between

shaped mass are numbers of large and small, rounded, oval and elongated faintly reddish, soft-glistening pedunculated masses. Owing to the disordered condition of the specimen, it is impossible to say whether these small masses arise from the mucous membrane or from the muscular coat.



FIG. 231 (A, *Top*) Hemangio-endothelioma of the stomach (B, *Bottom*) Appearance of the tumor

The report of the microscopic examination follows:

"Microscopic examination of celloidin section of one of these pedunculated masses shows the presence of a tumor, the unit of which is a small, thin-walled blood vessel imbedded in a more or less richly cellular collection of cells of indefinite histology. In view of the preponderance of blood vessels, the most appropriate diagnosis would appear to be that of hemangioma. However, in view of the uncertainty of diagnosis based on frozen sections, decision is postponed until celloidin sections are available.

"Microscopic examination of celloidin sections fails to show the same great abundance of blood vessels as revealed in the frozen section. On the other hand, the preponderating cell is a rather small, sharply outlined cell with a sharply limited membrane, a stainless cytoplasm, and a small, rounded or slightly oval, poorly chromatic nucleus. By far the greater part of these cells are arranged diffusely. Among them, in places, are rounded or oval or slitlike crevices, corresponding, apparently, to vessels. In still other places, arranged singly or in small groups, are small thin-walled vessels lined by endothelium and filled by red cells."

The diagnosis was stated as follows:

"Combining the appearances in the frozen





FIG 232 (A Left) Fibromyoma pars cardia of the stomach (B Right) Same patient Note the translucent area surrounded by barium in the pars cardia, produced by the tumor

and celloidin sections, a diagnosis of heman-
gio endothelioma. I think entirely justifi-
fiable.

Roentgen examination (Fig 231 A) re-
vealed the presence of a large well defined
translucent area in the pyloric portion of the
stomach with a complete absence of normal
mucosal markings. The lesser and greater
curvatures of the stomach of this region were
intact. There were no disturbance of gastric
peristalsis through this region. The roentgen
diagnosis was tumor involving the pyloric
portion of the stomach apparently benign. As
verified surgically this proved to be a heman-
gio endothelioma. The appearance of the
tumor is to be seen in Figure 231 B.

The next case is that of a fibromyoma
of the stomach.

E G female, aged 45. During the preced-
ing 5 months, the patient complained of
epigastric pain occasionally radiating to the
back and to the precordium. The pain was
worse from 1 to 3 hours after meals and was
relieved by eating. It occasionally woke her
from sleep. There was no vomiting, jaundice
or bleeding and no weight loss. A transtho-
racic resection was done and the proximal
portion of the stomach was removed for a
tumor present in the pars cardia. The distal

portion of the stomach was anastomosed to
the esophagus.

The report of the gross examination was:

There is a large, lobulated tumor mass in the
cardiac region measuring 10 x 5 x 5 cm. It is
mostly exogastric in location but also in-
volves the wall of the stomach and bulges into
the lumen. The mucosal surface over the
tumor shows a 1.5 cm ulceration. The part
surrounding this ulceration is fixed to the
tumor while the rest is freely movable over it.
The part of the tumor outside of the stomach
appears completely encapsulated. The surface
is yellowish red in color. The consistency is
moderately firm. On section the cut surface
consists of tannish white tissue, which shows
radial striations (Fig 232 A).

The final diagnosis was endoexogastric
fibromyoma of the stomach.

Roentgen examination (Fig 232 B) showed
a mass in the pars cardia encircled by barium.
A diagnosis of tumor of the pars cardia was
made. The tumor was considered to be prob-
ably malignant.

Rarely benign tumors of the stomach
may be multiple as illustrated by the next
case.

N D female, aged 70. During the preced-
ing 2 months the patient had complained of a
dull ache in the epigastrium occurring about



FIG 233 (A, *Left*) Multiple polyps of the stomach (B, *Center*) Same patient Note the small, rounded translucent areas in the pars media and in the pyloric antrum produced by the polyps (C, *Right*) Same patient Additional translucent areas produced by the polyps

2 hours after meals and lasting about 2 hours. The distress was relieved by milk of magnesia tablets. She vomited on one occasion. There was no blood in the vomitus nor was there any blood in the stool at any time. She had lost 20 pounds in 2 months. Physical examination of the abdomen was essentially negative except for some tenderness in the epigastrium.

At operation, a gastrotomy was done. Numerous polyps of the stomach were found, particularly in the pars media and on the lesser curvature. Very few of the polyps were in the pylorus. The polyps ranged in size from a few mm to 2 cm in diameter. Four polyps were removed by clamping the pedicle and amputating the polyps by sharp dissection. Many of the polyps, which were small in size, were left undisturbed.

The pathologic diagnosis was adenomatous polyp of the stomach.

Figure 233 A shows the appearance of the polyps removed at operation.

Roentgen examination (Fig 233 B) showed definite evidence of two small tumors, one hugging the lesser curvature, pars media, and one more centrally located. A number of translucent areas of variable size were present in the pars pylorica. Figure 233 C, in which the two tumors were obscured, showed another tumor near the greater curvature as well as vaguely outlined translucent areas in the pars pylorica. The preoperative diagnosis was multiple gastric polyposis.

The following case is another example of multiple polyps of the stomach and of the importance of proper technic in their demonstration.

C A, male, aged 58. The patient was on the psychiatric division of the hospital because of an undiagnosed psychosis. A good history of his complaints was not attainable. Physical examination and roentgenographic studies showed evidence of active pulmonary tuberculosis. Because of anemia and blood in the stools a roentgenographic examination of the gastro-intestinal tract was made. The findings in the examination of the stomach were significant (Fig 234 A and B). In Figure 234 A, representing the appearance when the stomach was completely filled with barium, there is no evidence of any abnormality. On examination with a thin layer of barium (Fig 234 B) many small, rounded translucent areas varying in size were found within the lumen of the stomach. Based on these findings a diagnosis of multiple polyps of the stomach was made.

At operation palpation of the stomach revealed several marblelike masses in its distal half. A subtotal gastrectomy was done.

Macroscopic examination of the resected specimen (Fig 234 C). "Specimen received fresh consists of a portion of stomach opened. There are five spheroid pedunculated reddish rather soft masses on the mucosal surface covering about 1.5 cm in diameter. In addition there are ten firmly slightly elevated nodules about 1 cm in diameter."

Microscopic Examination. "Sections reveal adenomatous polyps of varying size and shape. The smallest polyp is just a slight local thickening of the mucosa by regular mucosal cells. The larger polyps contain glands of varying size, some of which are dilated and contain faint basophilic material, while others are small and have vascular nuclei and scanty



FIG 234 (A *Top left*) Multiple polyps of the stomach not visualized because of overdistention with barium (B, *Top center*) Examination by the thin layer method. The many rounded translucent areas produced by the polyps are now visible (C, *Top, right*) Appearance of resected specimen showing the many polyps of the stomach

FIG 234 (D *Right*) Multiple polyps of the stomach distributed along the greater curvature (confirmed at autopsy). Note the rounded translucent areas along the greater curvature of the stomach produced by the polyps. When the stomach was distended with barium the translucent areas were obscured



cytoplasm. None of the polyps contains a fibrous stalk. One of the larger polyps is congested and contains scattered recent hemorrhage. There is a small amount of acute inflammation at the tip of the larger polyps. There is no invasion of the submucosa. The muscularis mucosa appears distorted and extends into the stalk of the polyp.

Diagnosis: Adenomatous polyp of the stomach.

This case then is of considerable interest not only because it is an example of multiple benign polyps of the stomach but also because of the technical features involved in the roentgenographic demonstration of the lesions. If numerous polyps of the stomach may be obscured when the stomach is distended with barium then obviously a single small polyp may go unrecognized unless the thin layer method is employed as part of a complete investigation.

The presence of multiple polyps of the stomach was confirmed by autopsy in the following case:

C-1, male, aged 57. The patient gave a

10 year history of recurring episodes of upper abdominal pain. The pain was epigastric in location, occasionally radiated to the back and was not definitely related to food intake. There was no vomiting or melena. On the day of admission to the hospital he coughed up some blood. During the week preceding hospitalization he became hoarse and had a dry cough. He lost 15 pounds in the preceding 2 months. Gastric analysis showed an achlorhydria.

Roentgenographic examination of the chest revealed evidence of a malignant tumor with metastases to the ribs. Physical examination of the abdomen was essentially negative except for tenderness in the epigastrium. Endoscopic examination revealed paralysis of the left vocal cord. Roentgenographic examination of the stomach (Fig 234 D) was reported as follows: The mucosal folds along the greater

curvature of the pars media are of increased prominence and have a polypoid appearance. Corroborative gastroscopy is suggested." The patient's condition became rapidly worse, and he expired without gastroscopy having been done.

Autopsy revealed a papillary adenocarcinoma of the bronchus in the left lower lobe with secondary adenocarcinoma in the mediastinal lymph nodes, lung, liver, adrenals, vertebrae, ribs and kidneys. The findings in

the examination of the stomach are of particular interest. "The stomach is thin-walled and contains about 300 cc of greenish fluid. The mucosa is pale, and the rugal pattern is very much smoothed out. About 10 small polyps, peanut sized, are seen along the greater curvature."

The rounded areas along the greater curvature of the stomach as noted in the roentgenogram were evidently then due to the polyps found at autopsy.

REFERENCES

- 1 Cruveilhier, J. Anatomie pathologique du corps humain, Paris, 1835-1842.
- 2 Chaput, H. Enorme adénome pédiculé de la paroi postérieure de l'estomac, Bull Soc Anat Paris 9 534, 1895.
- 3 Sherren, J. External polypoid tumors of the stomach, Brit M J 2 593, 1911.
- 4 Collier, W. Multiple polypi of stomach and intestine, Tr Path Soc London 47 46, 1896.
- 5 Versé, M. Über die Entstehung, den Bau und das Wachstum der Polypen, Adenome und Karzinome des Magendarmkanals, Arb d path Inst, Leipzig, 1, No 5, 1-175, 1908.
- 6 Menetrier, P. Des polyadénomes gastriques et leur rapports avec le cancer de l'estomac, Arch de physiol norm et path, 1 (ser 4) pp 32-55 and 236-262, 1888.
- 7 Klob, J. Pancreas-Anomalien, Ztschr Gesellsch d Ärzte, Wien, 15 732, 1859.
- 8 Evans, J. A., and Weintraub, Sydney. Accessory pancreatic tissue in the stomach wall, Am J Roentgenol 69 22, 1953.
- 9 Faust, D. B., and Mudgett, C. S. Aberrant pancreas, with review of the literature and report of a case, Ann Int Med 14 717, 1940.
- 10 Barbose, Jorge, de Castro, J., Dockerty, M. B., and Waugh, John M. Pancreatic heterotopia, Surg, Gynec & Obst 82 527, 1941.
- 11 Present, A. J. Aberrant pancreas, Am J Roentgenol 56 55, 1946.
- 12 Roach, J. F. and Poppel, M. H. The Roentgen demonstration of an aberrant pancreatic nodule in the stomach, Am J Roentgenol 56 586, 1946.
- 13 von Keiser, D. Das Nebenpankreas unter dem Bilde benignen Magentumoren, Chirurg 19 154, 1948.
- 14 Lapidari, Mario. Pancreas accessorio dello stomaco o metaplasia eterotopica a tipo pancreatico della mucosa gastrica, Arch ital chir 47 432, 1937.
- 15 Benner, W. H. Diagnostic morphology of aberrant pancreas of the stomach. Report of five cases, Surgery 29 170, 1951.
- 16 Joyeux, R., Guibert, H. L., and Guerrier. Les pancreas aberrants gastro-duodenaux, J chir 63 261, 1947.
- 16A Littner, M., and Kush, I. Aberrant pancreatic tissue in the gastric antrum, Radiology 59 201, 1952.
- 17 Branch, C. D., and Gross, R. E. Aberrant pancreatic tissue in the gastrointestinal tract, Arch Surg 31 200, 1935.
- 18 Wirts, C. W., and Breckenbridge, R. L. Carcinoid tumor (argentaffinoma) of the stomach, Gastroenterology 12 682, 1949.
- 19 Meyer, J. S. Polyposis gastrica (polyadenoma), JAMA 61 1960, 1913.
- 20 Balfour, D. C. Polyposis of the stomach, Surg, Gynec & Obst 28 465, 1919.
- 21 Ruggles, H. E. Unusual gastric polyp, Am J Roentgenol 7 356, 1920.
- 22 Rigler, L. G. Roentgen observation of benign tumor of stomach prolapsing through the pylorus. case report, Am J Roentgenol 20 529, 1928.
- 23 Wade, H. Intussusception of the stomach and duodenum due to a gastric polypus, Surg, Gynec, & Obst 17 181, 1913.
- 24 Lonnerblad, L. Zwei Fälle von Mageninvagination, Acta radiol 14 82, 1933.
- 25 Natorp, W. Gestielter Polyp des Magengewalbes, Röntgenpraxis 3 456, 1931.
- 26 Odquist, H. Beitrag zum Röntgenbild des Magenneurinoms, Acta radiol 18 112, 1937.
- 27 Renander, A. Gutartige Magentumoren, Acta radiol 17 491, 1936.

- 28 Senturia, H R Gastric neurinoma, Am J Roentgenol 43 61, 1940
- 29 Gomez J Schwannome de l'estomac J chir 63 463 1947
- 30 Ludin Max Endogastritisches Schwan nom Schweiz med Wchnschr 75 740, 1945
- 31 Jones, C K, and McKee, F W Gastric paraganglioma with ulceration report of a case, Arch Path 48 570, 1949
- 32 Paaby, H Benign tumors of the stom ach A case of lipoma submucosa ventri culi simulating cancer of the stomach, Acta chir scandinav 97 381 1949
- 33 Alvarez I F, Lastra J E and Leon P Ulcerated gastric lipoma Gastroenterol ogy 11 746, 1948
- 34 West, J P and Knox G Neurogenic tumors of the stomach, Surgerv 23 450 1948
- 35 Gentry, R W, Dockerty M B and Clagett O T Vascular malformation and vascular tumors of the gastrointes tinal tract Surg Gynec & Obst 88 281 1949
- 36 Heitz, J Liomyome calcine sans sereux de la paroi stomacale Bull et mem Soc anat Paris 77 896 1902
- 37 Meissner W A Leiomyoma of the stomach, Arch Path 38 207 1944
- 38 Garbarini, J, and Price H P Calcified leiomyoma of the stomach, New England J Med 243 406, 1950
- 39 Koloski E L, Schallenberger, P L and Hawk, G W Partially calcified gastric leiomyoma Am J Surg 80 245 1950
- 40 Leigh, T F Calcified gastric leiomyoma, Radiology 55 419 1950
- 41 Forssman G Die Roentgendiagnostik benigner Magentumoren Acta radiol 24 135, 1943
- 42 Enfield C A Pedunculated intragas tric tumor Am J Roentgenol 25 515 1931
- 43 Simonson M Zur Frage der gutartigen Magentumoren, Rontgenpraxis 6 663, 1934
- 44 Hollmann, W Magenkarzinom und Ma genpolyp, Rontgenpraxis 8 361, 1936
- 45 Miller T G Elason, F L, and Wright, V W M Carcinomatous degeneration of polyp of the stomach, Arch Int Med 46 841 1930
- 46 Benedict, E B and Allen, A W Ade nomatous polyp of the stomach with special reference to malignant degenera tion, Surg, Gynec & Obst 58 79, 1934
- 47 Vinnes J F, and Geschickter, C F Benign tumors of the stomach, Am J Cancer 28 136 1936
- 48 Brunn L and Pearl, H Multiple gas tric polyposis Surg, Gynec & Obst 76 257, 1943
- 49 Pendergrass E P Prolapse of pedun culated tumors and gastric mucosa through pylorus into duodenum roent genologic diagnosis, J A M A 94 317 1930
- 50 Manning I H Jr and Gunter J U Prolapse of redundant gastric mucosa through the pyloric canal into the duo denum, Am J Path 26 57 1950
- 51 Jeghers H McKusick V A and Katz k H Generalized intestinal polyposis and melanin spots of the oral mucosa lips and digits New England J Med 244 995 1949 244 1031 1949
- 52 Wolff, H H Familial intestinal polyp osis with pigmentation of lips oral mu cosa face and digits Lancet 1 446 1952
- 53 Ravitch M M Polypoid adenomatosis of the entire gastrointestinal tract, Ann Surg 128 283 1948
- 54 Welch, G, and McHardy, G Adenoma tous polyposis of the whole gastrointes tinal tract, Gastroenterology 13 451, 1949
- 55 Andrew, R Generalized intestinal poly posis with melanosis Gastroenterology 23 495, 1953

in Inlow's case showed a bezoar in the stomach which on removal was proved chemically to consist of shellac. The shellac had separated from the solution in denatured alcohol which the patient had been imbibing during the prohibition era. In all the cases reported in the literature, the cause was obviously the drinking of shellac solution in some form. In the majority of the cases there was a concomitant gastric ulcer. The ulcer developing in association with a bezoar may perforate.¹

Ramsbottom and Barclay¹³ made one of the earliest diagnoses of hair ball of the stomach by roentgen examination. The bismuth meal encompassed the mass, which produced a lighter area within the shadow of the bismuth. Palpation demonstrated that

the mass was freely movable within the stomach. Another case of hair ball of the stomach diagnosed roentgenologically was that of Rovsing.¹⁴ The patient was an 8 year old girl who was in the habit of eating her hair. The barium meal showed an irregularly outlined, movable, translucent mass of the shape of the stomach. At operation the roentgen diagnosis was confirmed, and a hair ball was removed.

There are three roentgenologic points which aid in its recognition: (1) the shadow produced by the mass is centrally located, so that the contours of the stomach are unaffected and exhibit normal peristalsis; (2) the intragastric position of the mass may be altered manually under fluoroscopic control; and (3) if the mass is large, it may in



FIG. 235 (A Top) Phytobezoar with gastric ulcer (B Bottom) Same patient. Appearance of the phytobezoar.

vaginate the air bubble of the stomach (in the erect position) and stand out in clear relief

The typical roentgen appearance of a large hair ball of the stomach was shown in the case of a little girl, 8 years old, described by Mercier and Mangini.¹⁵

Allen¹⁶ reported the roentgen findings in three cases of phytobezoar. In two cases they produced irregularly outlined trans-

lucent areas, exhibiting free mobility. In the third case the barium-impregnated phytobezoar could be distinguished from the rest of the barium in the stomach by a translucent halo. While the presence of the bezoar is ordinarily recognized radiologically without difficulty, at times it may closely simulate the deformity produced by an intrinsic polypoid mass.¹⁷

Rarely, a new growth may simulate the

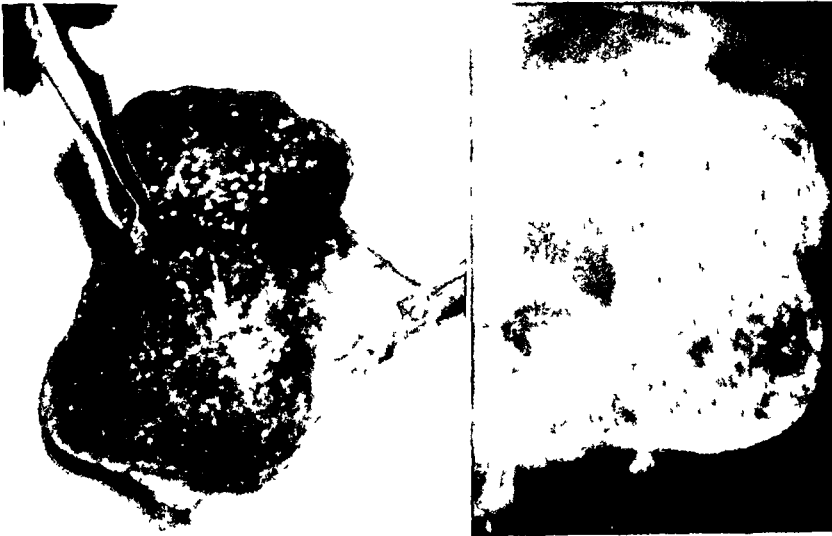


FIG 236 (A, *Left*) Trichobezoar of the stomach (B, *Right*) Same patient. A translucent area, occupying almost the entire stomach, was produced by the bezoar.



FIG 237 Mass of paper clips in the stomach

appearance of a bezoar Sanguily and Blanco¹⁸ described a huge intragastric mass which occupied almost the entire stomach, radiologically simulating the appearance of a bezoar Operation revealed a mass attached by a pedicle to the posterior wall of the fundus from which it extended into the gastric cavity the tumor having assumed the shape of the stomach itself Histologic examination revealed it to be a schwannoma

A myoma may be so huge and conform to the configuration of the stomach so closely as also to simulate the roentgen appearance of a bezoar¹⁹

Illustrative Cases The roentgen appearance produced by a phytobezoar of the stomach is well shown in the following case examined by Dr Lewis G Allen The irregularly outlined translucent area resulting from the presence of a phytobezoar can be noted in the accompanying illustration

(Fig 235 A) In addition, a large ulcer of the lesser curvature of the stomach was present Figure 235 B shows the appearance of the phytobezoar after removal

In the following case the trichobezoar occupied almost the entire stomach

C H, aged 4 At the age of 6 months, this patient of Dr Stillman's picked the wool off blankets and swallowed it The parents therefore found it necessary to cover the blankets with muslin Later, the child pulled out some of her hair and swallowed it, a habit which continued for about 6 months The hair was therefore clipped short during the next 6 months and then permitted to grow again At one time the child vomited a hair ball about the size of a hen's egg The child's habit continued and shortly before operation the mother noticed hair in the child's stool The child finally developed severe abdominal pain Operation under spinal anesthesia revealed a hair ball, which was removed The trichobezoar was hard and firm and had the consistency of thick felt (Fig 236 A) Roentgen



FIG 238 Stomach filled with tacks

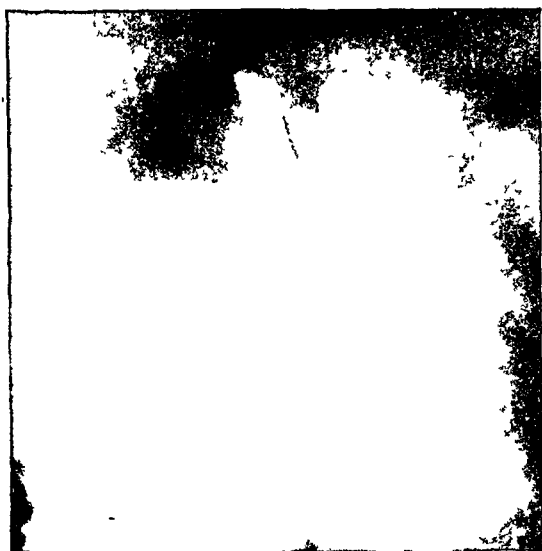


FIG 239 Spoon in the stomach

examination showed a large, freely movable mass, filling almost the entire lumen of the stomach (Fig 236 B)

Other foreign bodies may also be swallowed by patients, often with intent, as in the mentally unbalanced

An example of foreign bodies in the stomach is illustrated by Figure 237. The patient, a boy of 14 on the Psychiatric Service at Bellevue Hospital, was in the habit of swallowing all sorts of objects, such as buttons, nails and paper clips. Roentgen examination shows the numerous paper clips within the stomach. None was noted elsewhere in the alimentary tract.

Another example is illustrated by Figure 238, showing a stomach the lower portion of which is filled with tacks. A few have escaped beyond the confines of the stomach itself. Figure 239 shows a spoon in the stomach, which was removed by gastrotomy.

REFERENCES

- 1 Baudamant Description de deux cas, masses de cheveux trouvées dans l'estomac et les intestins d'un jeune garçon âgé de seize ans, *Hist de la Soc Roy de Méd*, Paris 2 262, 1777-1778

- 2 Butterworth, W W Hair ball or hair cast of the stomach and its occurrence in children report of two instances with bibliography and synopsis of cases reported to date, *J A M A* 53 616, 1909
- 3 Kampmann, E Ein Trichobezoar im Magen (Gastrotomie), *Munchen med Wchnschr* 58 413, 1911
- 4 Thorek, P, and Rutter, C Trichobezoar and phytobezoar, *Am J Surg* 35:603, 1937
- 5 Chont, L K . Phytobezoar and its formation in vitro, *Radiology* 38 14, 1942
- 6 Rodgers, F D Phytobezoar of persimmon origin, *Radiology* 29.494, 1937
- 7 Outten, W B A case of double gastrolith removed by gastrotomy, *M Fortnightly* 6 445, 1894
- 8 Davies, I J Hair ball or hair casts of the stomach and gastro-intestinal tract, *Lancet* 2 790, 1921.
- 9 DeBakey, M, and Ochsner, A Bezoars and concretions Comprehensive review of literature with analysis of 303 collected cases and presentation of 8 additional cases, *Surgery* 6 934, 1938
- 10 Satterfield, W T Case report, *Memphis Med* 12 140, 1937
- 11 Inlow, H Shellac bezoars, *Radiology* 34 618, 1940
- 12 Levy, J S, and Smith, R T Trichobezoar Report of a case, *Am J Digest Dis* 9 198, 1942
- 13 Ramsbottom, A, and Barclay, E The diagnosis of a hair ball in the stomach, *Arch Roentgen Ray* 18:167, 1909
- 14 Rovsing, C M Trichobezoars in the stomach and their demonstration by roentgen examination, *Acta radiol* 2 491, 1923
- 15 Mercier, R, and Mangini, P Egagropile volumineux de l'estomac, *Gaz méd de France, supp radiol* 43:35, 1936
- 16 Allen, L G Phytobezoar report of three cases, *Am J Roentgenol* 39 67, 1938
- 17 Droegemuller, E H Phytobezoar, *M Clin North America* 13 1539, 1930
- 18 Sanguily, J, and Blanco, F L Gastric schwannoma, *Surgery* 17 328, 1945
- 19 Husebye, O W Case of benign myoma in the stomach, *Acta radiol* 29 525, 1948

Malignant Tumors of the Stomach

INCIDENCE AND LOCATION

In the male, the stomach is the most common site for the development of cancer. Though a site of predilection also in the female, it is less common than cancer of the uterus.

In about 60 per cent of the cases the tumors occur in the pyloric portion of the stomach. Twenty per cent originate along the lesser curvature and cardia and about 5 per cent arise on the posterior wall. The rest are localized along the greater curvature, the anterior wall and the fundus. The least frequent area of involvement is the fundus. Adenocarcinoma is the most common type of malignancy of the stomach.

Malignant tumors of the stomach may be entirely asymptomatic and may be found only on accidental examination or the discovery of a metastatic focus. At times a gastric new growth is so free of subjective and objective evidence as to be discovered only at autopsy. They may be multiple.^{1,2} In the case reported by Rickles there were 5 independent carcinomas of the stomach.

ROENTGEN DIAGNOSIS

The importance of the roentgen method in the diagnosis of carcinoma of the stomach lies in the fact that the early clinical symptoms may be vague and indefinite. Roentgen examination may disclose evidence of serious abnormality long before the clinical picture would definitely justify the diagnosis of malignant disease.

Unfortunately, however, due to the very vagueness of the early symptoms patients

often do not present themselves until so late that roentgen examination reveals the malignant lesion to have reached an inoperable stage. This situation is complicated still further by the fact that even when the patient presents himself at the very onset of the disease, careful study may fail to demonstrate the lesion in its incipient stage. This is particularly true of the lesion which infiltrates the submucosa and for a long time may affect neither the external contour nor the intrinsic mucosal configuration.

An extremely important feature in relation to the roentgen diagnosis of the stomach when malignancy is suspected is the significance of a normal gastric appearance. When the examination has been conducted carefully, including observation through every angle of obliquity in both the vertical and the prone positions as well as a detailed study of the mucosal structure in the vast majority of cases normal findings will justify the diagnosis of the absence of malignant disease.

Another aid that can be obtained by roentgen study is the differentiation between an extragastric and an intragastric tumor. Thus the mere finding on physical examination of a palpable tumor in the gastric area in a patient assumed to have evidence of gastric disease does not necessarily prove beyond a doubt the exact anatomic location of the lesion. Fluoroscopic examination may disclose the fact that the contour of the stomach is intact and that peristalsis is normal. Moreover, the position of the stomach may be altered and the extragastric location of the tumor may be clearly shown by manual palpation.

Another aid in differential diagnosis lies in the demonstration of the mucosal "relief." This will be intact when the growth remains extragastric but may show areas of destruction when the growth is intrinsic.

A point of considerable practical clinical importance is the question of the operability of a tumor of the stomach. A malignant lesion which involves the entire stomach or one that is situated at the cardiac end may require a total gastrectomy. However, a tumor limited to the pyloric portion may be readily resectable. In addition to the actual localization of the growth, there are several other important points in this connection. First is the free mobility of the involved zone. The question of mobility of the stomach in the presence of malignant infiltration is, of course, extremely important from the standpoint of surgical intervention. The greater the mobility of the stomach, the less technical difficulty will the surgeon presumably encounter in an attempted resection. Another point of considerable practical significance is the fact that the size of the malignant infiltration offers no evidence as to the probable extent of metastatic involvement. Lesions of the stomach so small as to create the hope of a diagnosis at an early stage may show at operation widespread metastases. On the other hand, large lesions readily palpable may exhibit no evidence of the spread of the malignant lesion. The location of the tumor is much more important than its size in determining the question of operability.

Finally, in some cases roentgen study may disclose evidence of metastatic invasion which may escape the observation of even an excellent clinician. Thus, in addition to such clinical evidence as a Virchow's node, Blumer's shelf or fixation of the umbilicus, roentgen study may disclose invasion of the lungs and skeletal structures. Occasionally a marked delay in the passage of the barium through the transverse duodenum, with dilatation proximal to the zone of obstruction, in a patient with definite carcinoma of the stomach may suggest me-

tastases to retroperitoneal nodes as a cause of the occlusion. An examination of the colon may disclose the presence of a tumor invading the rectal wall, which may draw attention to a gastric malignancy as the primary cause.

In a small percentage of cases, the ovaries are involved by metastatic invasion. A diagnosis of a Krukenberg type of tumor may be justified by the roentgen demonstration of a gastric neoplasm, usually scirrhus, in a woman with ovarian enlargement. The involvement of the ovaries is apparently the result of peritoneal implantation and lymphatic spread.

The essential roentgen finding in malignancy of the stomach is the demonstration of a persistent defect of gastric contour, usually of irregular outline. In addition, frequently evidence of invasion of the lumen of the stomach may be shown. In order to demonstrate either or both of these findings, a careful, technically detailed study of every portion of the stomach is essential. The mucosal relief studies are, of course, of tremendous importance, yet these must not be pursued to the neglect of the study of the contour with the stomach completely filled with barium. The safest procedure and the maximum degree of exactness in diagnosis will be achieved through an intelligent combination of both these procedures. In many cases, a study of the contour of the filled stomach will be sufficient to establish the diagnosis, although the mucosal study even in such cases will yield more detailed information as to the extent of the growth. It is particularly in the case of malignant lesions that are ordinarily not recognizable by their involvement of either the lesser or the greater curvatures of the stomach that studies of the mucosa are of greatest value. In such cases a lesion on the anterior or posterior wall may actually be obscured when the stomach contains considerable barium. A small amount of a thin mixture, however, carefully spread over the entire mucosa, may disclose destructive changes arising from malignant invasion.

It is in such cases also that the compression technic popularized by Berg, Akerlund and Chaoul finds its greatest usefulness.

In the case of carcinoma at the cardiac end of the stomach, sometimes the diagnosis may be made on direct fluoroscopic inspection with the patient in the erect position. In such cases the soft tissue mass of the tumor may be outlined clearly as it extends within the air bubble. Inspection of the contour of the air bubble is, therefore, of considerable importance in the routine roentgen study of the stomach. This is especially so since barium administered to the patient may sink below the level of the tumor and show a normal contour. Another reason for careful fluoroscopic study in such cases lies in the fact that if the barium is observed leaving the esophagus a splitting of the stream of barium may be observed due to the interposition of the growth. Particularly when the tumor is small, such evidence of a persistent character of the splitting of the stream may assume enormous diagnostic importance. In addition, a careful study of this region will disclose the absence of normal mucosal markings. Films taken only in the prone position may fail to disclose a small tumor of the cardia because of the fact that it may be obscured by the upward displacement of the barium.

When the cardiac end of the esophagus is involved in the malignant process irregularities of contour, destruction of normal mucosal markings of the cardiac end of the esophagus and dilatation due to partial obstruction may be found, similar to the evidence produced when carcinoma is limited to the esophagus itself.

Of outstanding importance in the diagnosis of an infiltrating lesion of the stomach is the defect of contour usually irregular in outline. Such defects of contour may be simulated by a number of conditions. A small amount of barium, particularly when not uniformly distributed may produce apparent filling defects. These, however are of an inconstant character and will not be demonstrable on re examination. This is

one of the dangers in the application of mucosal studies when employed by the inexperienced. Retained food, as in pyloric stenosis, or improper preparation of the patient, with examination shortly after a meal may show abnormalities in contour which may suggest momentarily the presence of serious disease. However, the mobility of the contents, the demonstration of the inconstant nature of the deformity as well as questioning the patient will disclose the true nature of the condition.

The pressure of neighboring organs may also produce apparent deformities of contour. The pressure of the ribs against the greater curvature as well as the pressure of the spine may alter the contour of the stomach. Changing the position of the patient, the persistence of normal peristaltic activity, the effect of manual palpation in forcing barium into the apparently defective areas as well as the normal mucosal relief, all combine to make differential diagnosis easily possible.

Similarly displacements and indentations produced by extragastric tumors may be equally amenable to differentiation from intrinsic disease by the application of the procedure described above. The difficulty is much greater, however, when the stomach is embedded in extragastric tumefaction. In extreme cases of this character, peristaltic activity may not be readily demonstrable and the contour may be superficially irregular. Careful mucosal studies, exhibiting an unbroken membrane, may be the only criterion upon which a diagnosis of the extragastric location of the tumor may be based. When a carcinoma of the pancreas actually breaks through the wall and invades the lumen of the stomach the differential diagnosis may be difficult or impossible.

Benign lesions may produce deformities which may closely mimic those of malignant disease. Among these are lesions which have been classed as syphilis or tuberculosis of the stomach, benign hypertrophic pyloric stenosis, and inflammatory lesions produced by the swallowing of corrosives.

At times, only a very careful clinical investigation of the entire background of the case will aid in determining the etiologic factor. Roentgenologically, however, the differentiation can be made only with extreme difficulty, and in some cases it may be impossible definitely to evaluate the significance of the deformity.

The difficulty is enhanced still further by the fact that, in the early stages of scirrhus carcinoma, the pathologic process which is essentially limited to the submucosa and the deeper layers of the stomach may leave the mucous membrane intact and free of any roentgenologically recognizable abnormalities.

Spasm of various regions of the stomach may occasionally introduce difficulties in differential diagnosis. However, deformities produced in this manner will, as a rule, be inconstant, either during the course of a single fluoroscopic examination or on repeated studies with and without the preliminary administration of antispasmodics.

Occasionally, also, a marked hypertrophic gastritis with considerable distortion of the mucosal relief picture may introduce difficulties in differential diagnosis. A careful evaluation of the appearance of the mucosal folds will be necessary. The continuity of the hypertrophic folds with the rest of the folds of the stomach that are uninvolved may aid in establishing the nature of the process as benign rather than malignant. Submucosal infiltration of a malignant character may introduce difficulties which may make a differential diagnosis impossible.

The infiltrative type of new growth may give rise to considerable difficulty in two ways. In the first place, by extending beneath the mucosa, its recognition in the early stages may be difficult or impossible. While a mucosal fold may be thickened and therefore perhaps may become more prominent in the roentgen relief, it may not be possible to differentiate such an appearance from that of a prominent fold due either to functional causes or to a hypertrophic gastritis of benign origin. Even with

regard to the possibility of early diagnosis of carcinoma by gastroscopy, it may not always be possible to differentiate malignant infiltration of the wall from hypertrophic gastritis. Moreover, even when sufficient change has been produced by the infiltrative process so as to be recognizable or suspected, the exact limitations of its spread may be impossible to determine. This is true not only from the roentgen point of view, but even at the time of operation palpation may fail to give a clue as to its exact limitations.

Persistent irregularities of contour in occasional areas of the stomach, however, even though superficial in nature and associated with an absence of peristaltic activity in those regions, are an aid in the diagnosis of malignant disease.

Scirrhus carcinoma in its later stages produces a narrowed tubular stomach when the pathologic process is diffuse. Scirrhus malignant invasion of the stomach, however, may show a predilection for special areas. Thus, the process may be limited to the midportion of the stomach, leaving the cardia and the pylorus free of involvement. Owing to the narrowing of the pars media, the stomach becomes dumbbell-shaped or hourglass in appearance. The contour often exhibits superficial irregularities, although at times scirrhus invasion may leave the affected area surprisingly smooth. This type of organic hourglass deformity is readily distinguished from the type produced by an ulcer of the lesser curvature. Unlike the dumbbell-shaped appearance, the organic hourglass deformity of ulcer is B-shaped, with the lesser curvature uninvolved except for evidence of a niche. In addition, there are those other points in differential diagnosis which have been discussed previously under the subject of ulcer.

Owing to the stiffness and rigidity of the wall produced by the infiltrative process, the barium runs through this region with considerable rapidity. Despite this tendency, the upper locule in the late stages of emptying may show a moderate retardation. Par-

ticularly significant is the fact that the esophagus may remain filled because of the diminished capacity of the stomach. This is clinically important since under such conditions a patient may give a history of difficulty in swallowing and the impression that the cause is a malignant lesion of the esophagus itself. If the roentgen examination then is limited only to the esophagus, the exact nature of the cause may escape recognition.

When the infiltrative lesion is limited to the pylorus, pyloric stenosis may result, with marked dilatation of the stomach, an obstructive type of peristaltic activity and considerable gastric retention. In some cases the roentgen picture may be indistinguishable from the pyloric stenosis produced by an ulcer. It is essential to make every effort to demonstrate the entire contour of the lesser and the greater curvature. An unbroken contour with peristaltic activity traced to the pyloric ring would favor the benign nature of the obstructive process. An irregularity in the prepyloric region would speak for malignancy. All the other possibilities, however, of hypertrophic pyloric stenosis, syphilis or tuberculosis and chronic gastritis would have to be considered in arriving at a differential diagnosis. In those cases in which the infiltration at the pylorus results in rigidity and stiffness of the walls without occlusion there may be acceleration in the emptying of the stomach, the barium rapidly filling the duodenum and the rest of the small intestine.

The persistent absence of peristaltic activity at any localized region of the stomach is a matter of extreme diagnostic importance. This is particularly significant when associated with a definite defect in the contour. However, small localized areas of rigidity at the lesser curvature with smooth contour and an absence of peristalsis may be noted sometimes at the site of a mucosal ulcer or as a result of retrogressive changes in the healing of an ulcer. If the localized rigid area is produced by a small malignant

infiltration, the contour may show a superficial irregularity. More extensive evidence of destruction associated with this superficial irregularity may be demonstrable by mucosal studies and may aid materially in establishing the diagnosis. The palpation of the mass, the position of which coincides with the defect of contour, and the absence of peristalsis give additional confirmation to the diagnosis of malignant infiltration. Of least importance in the diagnosis is the demonstration of localized tenderness.

Disturbances of mobility in the presence of malignant infiltration may also be roentgenologically demonstrable, particularly by palpation during fluoroscopic examination. The tumor may be fixed to the liver as a result of malignant infiltration in the ligaments. Following metastatic spread in the mesocolon, the transverse colon may become closely attached to the stomach. There may, in fact, be an actual coalescence of the walls of the stomach and the colon, and, as the result of ulceration, a gastrocolic fistula may be established. This may then be recognizable, either by the ingested meal showing barium directly entering the colon through the stomach, or the fistulous communication may be clearly demonstrated by means of a barium enema.

In addition to the scirrhus type of malignancy, which is recognizable primarily by infiltration of the wall, there is the adenomatous or papillomatous type of malignancy which primarily invades the lumen of the stomach. This type of malignancy produces irregularly outlined defects within the lumen of the stomach by displacement of the barium. It is in malignant tumors of this kind that the fingerprinting type of deformity is found. Not only should examination be made with the stomach completely filled out but also with a thin layer of barium. At times large amounts of barium may obscure the presence of a tumor of this nature. However, if a small amount of barium is used and if this is carefully spread over the mucous membrane, the tumor mass will stand out in clear relief. Manual or

mechanical compression may also be of considerable importance. As with every other type of gastric lesion, here, too, examination in every position and through every angle of obliquity is important. Considerable care, of course, is required when the tumor is on the posterior wall.

Sarcoma of the Stomach Sarcomas of the stomach produce deformities in the roentgenogram indistinguishable from those caused by malignant infiltration generally. In rare cases the roentgen findings may be those of a benign tumor.^{4,5}

Lymphogranuloma and Leukemic Infiltration of the Stomach A detailed survey of the pathologic changes of the alimentary tract as the result of leukemic infiltration was made by Symmers⁶ at Bellevue Hospital. The gastric rugae were very prominent and, between the folds, the mucosa showed the presence of many isolated nodular masses, particularly in the pyloric region. These nodular areas resulted from collections of lymphoid tissue in the mucosa and submucosa.

The great thickening of the gastric rugae throughout the entire stomach as a result of leukemic infiltration was also described by Wells and Maver.⁷ This finding was true not only in their personal case, but also in the 6 out of 7 cases of pseudoleukemic infiltration which they found in the literature.

Pearson, Stasney and Pizzolato⁸ described the huge, irregular folds of the stomach when involved in lymphatic leukemia. These were most marked on the greater curvature toward the pylorus. Pathologically, the process was limited to the mucosa and the submucosa, with sharp demarcation from the muscularis. In myelogenous leukemia, however, they noted infiltration of the deeper layers. Therefore, the roentgen picture may be that of a polypoid hypertrophic gastritis, and only histologic study will demonstrate the true nature of the disease.^{9,10}

A feature in the roentgenographic study in such cases that may be helpful depends on the fact that the duodenum may be seri-

ously involved in the leukemic infiltrative process as well as the stomach. Therefore, roentgenographic study may show unusual prominence of the mucosal relief, with polypoid changes in the duodenum, as described by Mead¹⁰ and Koch.¹¹ Thus, in Mead's case of chronic lymphatic leukemia, the involvement of the stomach was shown roentgenologically by unusual prominence and irregularity of the mucosal relief of the entire stomach, from cardia to pylorus, the appearance suggesting a severe gastritis or diffuse polyposis. A similar appearance was noted in the duodenum. Operation revealed the stomach to be markedly dilated and apparently filled with small tumors. The first and the second portions of the duodenum were also involved in the process, as well as areas throughout the remainder of the intestine. Examination of two lymph nodes near the stomach showed evidence of lymphatic leukemia on microscopic examination.

Similarly, Koch described two cases in which, in addition to marked thickening of the mucosal folds of the stomach, irregularly outlined translucent areas involved the pylorus and extended into the proximal portion of the duodenum.

The development of niche formation of the lesser curvature with associated irregularity of the pylorus as the result of lymphogranulomatous infiltration of the stomach was described by Kaznelson.¹²

In Hodgkin's disease of the stomach, the roentgenologic findings have been indistinguishable from those produced by malignant infiltration (Singer,¹³ Koenig and Culver,¹⁴ Jungmann,¹⁵ Pirkey and Roberts¹⁶ and Sandwick¹⁷). Singer's well-supported case was subjected to gross and microscopic study of the tissues at autopsy, and in this way he was able to demonstrate that the disease was limited to the stomach and the perigastric lymph nodes. The roentgenogram showed a deformity of the pylorus similar to that produced by a carcinomatous lesion. The only method of establishing the diagnosis is histologic examination, and even

then considerable difficulty may be encountered. Wallhauser¹⁸ has described the confusion in terminology and has shown that there are 52 different names for apparently the same disorder, some of the terms ranging from "chronic benign lymphomatosis to lymphosarcoma of the Hodgkin type." This is due to the fact that the criteria necessary to establish a diagnosis of Hodgkin's disease and many neoplastic structures of lymphoid origin have been grouped by some writers under the unqualified term "lymphoblastoma." In a lesion, therefore, in which even the histologic criteria are sometimes in doubt, one can hardly expect that roentgenologic examination will offer aid in differential diagnosis.

While the differential diagnosis of leukemic infiltration from a malignant lesion may be impossible, roentgen evidence that may aid in arousing suspicion as to the presence of the disease is the combination of giant rugae localized primarily to the pylorus, and an associated involvement of the duodenum. Since malignancy of the stomach practically never extends beyond the stomach through the pyloric ring with any considerable invasion of the duodenum, such evidence in the roentgenogram should prove to be of value in the differentiation of a malignant lesion of the stomach from that produced by leukemic infiltration.

The roentgen findings in lymphoblastoma of the stomach are practically indistinguishable from those of gastric carcinoma, not only in my experience but in that of other observers.^{19, 20}

A plasma cell tumor of the stomach (Ende²¹) may produce deformities, indicating only that an infiltrative lesion is present without any specific diagnostic features. This was also true of the benign teratoma of the stomach in an infant described by Large, McChord and Neel.²²

AMYLOIDOSIS OF THE STOMACH

Steinhaus³ in 1902 described the findings in a patient who died following a massive

gastrointestinal hemorrhage. Autopsy revealed a combination of amyloid and hyaline infiltration of the heart, the stomach and the intestine without any recognizable etiology. The stomach was markedly dilated, and the lumen of the pylorus was abnormally constricted. There were no definite ulcerations, although in some areas the mucosa was defective. The wall of the pylorus was thickened. A number of additional reports followed (Beneke and Bonning in 1908, Schiller in 1909, Beclert in 1918 and Lubarsch in 1929, quoted by Clausen⁴). Koletsky and Stecher in 1939 made a thorough survey of the literature on primary systemic amyloidosis. They stated that Ritter in 1908 had also described a case of amyloidosis with involvement of the stomach.

Amyloidosis of the stomach may cause roentgenographic changes closely simulating those of an infiltrative lesion. Apparently the first roentgenographic evidence appeared in the article by Gottron⁶ published in 1932. The stomach was smooth in outline, peristalsis was superficial, and there was a marked delay in the visualization of the pylorus. When the pylorus was finally visualized, the folds appeared to be unusually prominent. The evidence was considered suggestive of an intramural lesion. Pathologic examination disclosed amyloid deposits in the stomach as part of a generalized process. A roentgenographic deformity of the pylorus having the appearance of an infiltrative lesion was described by Clausen in 1935 (*ibid.*) in a case of amyloidosis of the stomach confirmed by autopsy.

In another case of amyloidosis of the stomach Abner Golden⁷ in 1945 described irregular narrowing of the pyloric antrum and a 50 per cent gastric retention at 6 hours (the roentgenograms were not included). At operation a mass was palpated in the pylorus which was resected. On opening the stomach two superficial ulcerations of the mucosa were present. There was an increase of the interstitial tissues caused by the deposit of amyloid resulting in

atrophy of the muscle fibers. There were also slight amyloid deposits in the walls of the blood vessels. Fibrotic changes were present in the region of the ulcers. Amyloidosis of the stomach may also cause roentgenographic changes similar to those in hypertrophic gastritis (Shippo and Brannan²⁸).

NEUROFIBROMA OF THE STOMACH

A rare lesion of the stomach is neurofibroma. Von Recklinghausen,²⁹ in his classic on neurofibromatosis published in 1882, described the changes which he found at autopsy in the stomach and the intestine. Pathologically, the lesions were similar to those in the nodules of the skin.

Unless the deformity of the stomach occurs in the presence of von Recklinghausen's disease it has no distinguishing features which would justify a diagnosis of neurofibroma. In Bank's¹⁰ case of a neurofibroma of the stomach the deformity was similar to that of a carcinoma of the pylorus. Simulation of a malignant ulcerating lesion characterized the roentgenographic appearance of the cases reported by Baty³¹ and Smith.³² In Bock's³³ case roentgen examination showed a deformity of the pylorus with narrowing of the lumen in a patient who had von Recklinghausen's disease. The author does not state that this association had aroused any preoperative suspicion of a neurofibroma of the stomach, which the lesion proved to be.

In this regard Canney's³⁴ experience is very interesting. One tumor of the stomach which he resected proved to be a neurofibroma. There was no evidence of von Recklinghausen's disease. In another patient a neurofibroma of the stomach was removed, and the pathologic diagnosis was made that the lesion was probably a local manifestation of von Recklinghausen's neurofibromatosis. However, there was no evidence of any generalized process, at the time of operation. Eleven years later, although the patient had no symptoms referable to the original gastric lesion, the chest, the shoulders and the back were studded with typical

nodules of von Recklinghausen's disease. The patient stated that these lesions began to appear shortly after his operation.

Gillespie³⁵ described a case of multiple cutaneous neurofibromata associated with a large abdominal mass. Roentgenographic examination showed considerable deformity of the stomach, indicative of neoplastic infiltration. Histologic study of the gastric tumor revealed it to be a neuro-epithelioma. However, microscopic examination of one of the cutaneous lesions showed the typical appearance of neurofibroma as seen in von Recklinghausen's disease. In Truglio's³⁶ case of generalized neurofibromatosis, autopsy revealed a chronic stenosing gastric ulcer at the floor of which was a neurofibromatous node. In addition there were some neurofibromatous nodules at the base of some ulcers of recent origin in the anastomosed loop of small intestine.

In one case of neurofibroma which I examined, the roentgenographic appearance of the stomach produced by the lesion was indistinguishable from an infiltrating carcinoma. This case is described in detail at the end of the chapter.

In extremely rare instances Boeck's sarcoid may involve the stomach and produce a deformity roentgenologically indistinguishable from malignant infiltration (McKusick^{36A}).

Essential Roentgen Findings The essential findings in the roentgen diagnosis of malignant infiltration of the stomach are

1. An irregular filling defect in the contour of the stomach, of a persistent nature and unaffected by re-examination or by the administration of antispasmodics.

2. Absence of peristalsis throughout the area of involvement.

3. Rigidity of the involved area, with diminished flexibility due to the thickening of the wall by the infiltrating process.

4. When the infiltration of the wall is marked, the lumen of the stomach becomes considerably narrowed, and in diffuse scirrhous infiltration, the obliteration of the lumen may be extreme.

5. Abnormalities of the normal mucosal

relief pattern produced by the infiltrating process

6 Irregularly outlined translucent areas within the confines of the stomach, occasionally of the finger printing type, due to the intraluminal invasion in cases of fungating or polypoid malignant new growths

7 Diminution of mobility, this occurring when the tumor is fixed to adjacent structures

8 Disturbances of motility, depending upon the localization and nature of the infiltrating process so that when the walls of the stomach are rigid and the pyloric lumen is gaping, the escape of barium may be extraordinarily rapid. When the pyloric outlet is occluded, every gradation of gastric enlargement and abnormal retention may develop, depending upon the degree and the unyielding character of the obstruction which the lesion has produced

MASS ROENTGENOGRAPHY

In order to make it possible to diagnose carcinoma in its earliest stages, mass roentgenography has been recommended a procedure which has proved to be quite successful in the demonstration of unsuspected lesions of the chest. Theoretically at least, mass roentgenography would appear to be an ideal procedure in detecting unsuspected pathology. Nevertheless, it is interesting to note some of the results in the actual application of such a mass study by St. John, Swenson and Harvey.³⁷ They examined 2,432 persons of whom 491 were re-examined one year or more later. All of the patients were over 50 years of age. Although an attempt was made to include only those without digestive symptoms, they found it impossible to limit this group with accuracy because only a few people were entirely free of digestive symptoms. The examination was conducted essentially by rapid fluoroscopy with rarely more than a minute devoted to each patient. Of the total number of examinations they found two patients who had a carcinoma of the stomach—a ratio of 1:24 per thousand studied.

Is mass radiography of the digestive tract

of symptom free patients a practical procedure? The difficulties involved at the present time are almost insurmountable.

1 Even in the group examined by St. John and his associates, the number of cases with a malignant lesion of the stomach was remarkably small, in spite of the fact that the individuals were all over 50 years of age and many of them apparently not altogether symptom free.

2 If the procedure is to be really inclusive, then other age groups must also be included—certainly those beyond the age of 40 and to be really thorough perhaps it should include all those beyond the age of 30. The number of individuals who would then fall into this category would be so enormous as to make such a study a practical impossibility.

3 Since the purpose of such mass examination is to locate lesions in their earliest stages, then fluoroscopy alone is not sufficient. Even the highly trained fluoroscopist (and not all those who might be expected to participate in such a vast campaign would be peculiarly gifted) must admit that small lesions may escape his attention. Careful roentgenography will minimize his failures. All of us have had the experience of seeing deformities in the film which had not been suspected during fluoroscopic examination. Probably this will be particularly true when very large numbers of patients are examined because of the fatigue which may develop toward the end as well as the rapid manner in which the investigation is conducted. Since it is the early lesion such as the small polyp which we are most concerned to demonstrate, contour roentgenography and mucosal relief studies are essential. To pick out only those cases which show fairly gross evidence of deformity for later detailed study will solve only a small part of the problem. Therefore it is the very case that appears to be fluoroscopically normal that requires detailed study rather than the one which shows obvious evidence of the lesion. If we are to be of maximum benefit in the cure of the disease

4 Moreover, if routine examination of the stomach is important, why not also include the colon and perhaps the rest of the digestive tract? A carcinoma of the colon is as significant a lesion as a carcinoma of the stomach. Detection of the asymptomatic polyp of the colon on routine examination may prove to be particularly useful, since its removal may prevent the ultimate development of a carcinoma. As in the case of the stomach, however, a more meticulous technique may be required for the demonstration of the small polyp than the full-blown lesion which may have advanced beyond the stage of curability.

5 Another problem that still further complicates the situation and makes the approach to successful mass roentgenography even more difficult is the matter of how often to repeat these studies. The absence of any organic disease at the time of examination obviously gives no assurance as to the future. Therefore, the practical difficulties are enhanced enormously by the necessity of repeated surveys made at comparatively brief intervals of from 3 to 6 months.

6 In spite of all these objections more frequent roentgenographic examinations of the digestive tract are indicated.

A In the presence of digestive symptoms even though quite mild.

B In the presence of any unexplained anemia.

C This is particularly true in the case of pernicious anemia. The association of carcinoma of the stomach and pernicious anemia has long been recognized. Quincke, in 1876, is credited with having first described the association of these two lesions. From then on the literature records many such reports of the coexistence of both of these lesions. Moreover, this association was not a mere coincidence. Careful investigation showed that there was a definite predilection of the pernicious anemia patient to the development of carcinoma of the stomach (Rambach,³⁸ Zancan,³⁹ Doehring and

Eusterman⁴⁰). Cotti,⁴¹ in a remarkably complete survey of the literature on the subject, came to the conclusion that the frequent association of the two diseases was far from accidental, and that there is a definite predisposition of the pernicious anemia patient to develop carcinoma of the stomach.

Therefore, the importance of making roentgenograms of the stomach routinely in every case of pernicious anemia as emphasized by Rigler⁴² and his associates is quite obvious. Moreover, even the patient apparently cured of pernicious anemia is more susceptible to the development of carcinoma of the stomach, and repeated examinations, perhaps once every 6 months, may justifiably be made for an indefinite period of time.

D Another important group that might well be subjected to careful frequent roentgenographic examinations of the stomach is that comprising the blood relatives of a patient with pernicious anemia. Weinberg⁴³ emphasized the hereditary factor in pernicious anemia. This observation has been amply confirmed (Meulengracht,⁴⁴ Gram,⁴⁵ Conner,⁴⁶ Thiele,⁴⁷ Zancan and Cotti). Zancan stated that in 74 individuals who were blood relatives of patients suffering from pernicious anemia, he found 6 cases of carcinoma of the stomach (8%). On the other hand, in 85 blood relatives of patients who had carcinoma of the stomach only 3 were similarly afflicted (3.5%). This indicated the greater susceptibility of those in the first group.

Therefore, these blood relatives might be studied routinely from three standpoints: the presence of achylia gastrica, of pernicious anemia and of gastric carcinoma.

E Still another group that might justify routine periodic roentgenographic studies of the stomach are those with gastric anacidity. Bloomfield and Pollard⁴⁸ were quite optimistic, however, about the prognosis in unexplained gastric anacidity. In a group of 43 apparently healthy people with a



FIG 240 Carcinoma of the pylorus
Note the irregularly outlined translucent areas produced by the tumor

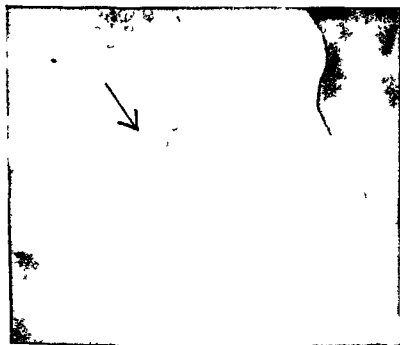


FIG 241 (A, *Top*) Appearance of a carcinoma of the stomach (B *Bottom*) Same patient Adenocarcinoma of the stomach Note the translucent areas within the stomach produced by the tumor

histamine refractory anacidity observed for a number of years not one developed carcinoma or pernicious anemia. Only 6 of the entire group were under the age of 40. They concluded that although their study did not preclude the possibility that gastric anacidity might be a precursor of carcinoma of the

stomach or pernicious anemia, the hazard to the individual appeared to be quite small. Conner⁴⁶ stated that achlorhydria may precede the development of pernicious anemia. In one case the disease occurred 12 years after the achlorhydria was discovered; in another case, 18 years later.

Perhaps patients with anacidity might be given precedence in the matter of routine roentgenographic investigation over those who are not only asymptomatic but also exhibit an essentially normal acid gastric secretion

Illustrative Cases The following cases illustrate the various roentgen features of gastric malignancy

A characteristic example of the fingerprinting type of deformity produced by an adenocarcinoma is shown in the following case

M. M., female, aged 65 For 3 years the patient noticed increasing weakness Nine months before her admission to the hospital, she developed pain in the epigastrium occurring 1 hour after meals, not relieved by food or medication, and occasionally lasting throughout the night There was no vomiting or bleeding There was a progressive pallor Physical examination disclosed an irregular, firm mass the size of a fist in the umbilical region

At operation a firm, irregular mass was found on the lesser curvature, pars pylorica which was resected

Pathologic examination of the specimen revealed adenocarcinoma of the stomach

Roentgen examination (Fig 240) revealed

changes characteristic of carcinoma of the pars pylorica, with deformity of the lesser curvature in this region and irregularly rounded translucent areas within the lumen

An example of the papillary adenomatous type of tumefaction is illustrated by the next case

W. R., aged 60 This patient gave a 4-week history of gnawing epigastric distress radiating throughout the entire abdomen and relieved for a few hours after eating There was no vomiting or evidence of bleeding Physical examination was essentially negative except for epigastric tenderness

The report at operation was as follows "There is a large, globular, cauliflower growth on the posterior wall of the stomach close to the lesser curvature (Fig 241 A) There is a circular pedicle 5 cm in diameter There is no infiltration of the wall surrounding the pedicle The latter is obscured by the main mass, which overhangs it at all points A few hard glands, 1 cm to 2 cm in diameter, are present in the lesser and greater omentum The pylorus and the cardiac end of the stomach are uninvolved"

Microscopic examination revealed papillary adenocarcinoma

Roentgen examination showed a large, irregularly outlined translucent area extending intraluminally from the lesser curvature, pars

FIG 242 (A, *Left*) Note the "fingerprinting" deformity of the stomach produced by the papillary adenocarcinoma of the stomach (B, *Right*) Gross appearance of the resected tumor The displacement of the barium by the cauliflowerlike tumor readily explains the deformities in the roentgenographic appearance of the stomach



media. In addition to the main defect there were several smaller areas of "fingerprinting," the entire appearance giving the impression of a cauliflowerlike tumor of the stomach originating from the lesser curvature (Fig 241 B). A comparative study of the roentgenogram and the specimen disclosed the anatomic reason for the particular roentgen deformity of the stomach.

Correlation of the underlying pathology with the roentgen appearance of a papillary adenocarcinoma of the stomach is illustrated by the following case.

M. A male, aged 69. One year prior to admission the patient had attacks of weakness which became progressive so that 6 months later he was no longer able to work. This was accompanied by marked anorexia and shortly before admission to the hospital he fainted on several occasions remaining unconscious for at least several minutes. Pain was present at times in the left upper quadrant. On physical examination a mass was palpable in the left upper quadrant. On proctoscopic examination a polyp was noted which was reported pathologically as a villous adenoma.

Exploration of the abdomen revealed a large firm irregular tumor mass within the

stomach. There were multiple pea and bean sized nodes in the gastocolic ligament and the gastrohepatic ligament. These were soft rubbery, smooth and reddened and gave the clinical appearance of inflammatory adenitis rather than tumor metastases.

A subtotal gastrectomy was done. Gross examination of the specimen was reported as follows: 'Within the stomach attached to the anterior wall adjacent to the lesser curvature by a stalk 5 cm in diameter is a mass measuring 15 x 10 cm and 4 cm thick. The mass is grayish white and somewhat friable. The tumor infiltrates the stomach wall at the base of the stalk. The remainder of the mucosa is normal. Omentum measuring 5 cm is attached. Five lymph nodes are identified all of which are along the greater curvature. One of these is white and firm.'

Microscopic Examination: "Papillary adenocarcinoma of the stomach. The regional lymph nodes have a chronic inflammatory reaction but no tumor cells are seen."

Roentgenographic examination (Fig 242 A) shows the fingerprinting defect of the stomach produced by the papillary adenocarcinoma. Figure 242 B shows the gross appearance of the papillary adenocarcinoma. Note how readily the abnormalities in the roentgen



FIG. 243 Adenocarcinoma of the stomach

ographic appearance of the stomach may be explained when comparison is made with the gross appearance of the tumor

At times, a proliferative, adenomatous, malignant mass may produce one large filling defect of the stomach with irregularity or absence of the gastric contour at the site of the new growth

O W, male, aged 57 This patient had been perfectly well until 1 year previously when he developed diarrhea, with from 6 to 10 loose bowel movements a day No blood or pus was present in the stool After 3 months the diarrhea subsided, and the patient began to experience epigastric pain, this was burning, came on from 20 to 30 minutes after the ingestion of food and occasionally was relieved by soda. He lost 25 pounds in weight Physical examination of the abdomen was essentially negative

Operation revealed a mass, 4 inches in diameter, involving the distal third of the stomach but not involving the pyloric ring There were large, hard nodes in the gastro-hepatic omentum, about the head of the pancreas and along the course of the aorta as far as the bifurcation The abdomen contained a small quantity of clear fluid The liver was

adherent to the diaphragm No nodules were present in the liver

At autopsy, at the pyloric end involving that structure on the superior curvature of the stomach there was a large, soft, circumscribed fungating mass, about the size of a lemon and projecting into the lumen, which so encroached upon the pyloric sphincter that only an orifice large enough to admit a probe remained Microscopic examination revealed adenocarcinoma of the stomach

Roentgen examination (Fig 243) revealed the presence of a large defect involving practically the entire pars pylorica There was complete absence of the greater curvature in this region The lesser curvature was slightly irregular The zone of invasion encroached upon the immediate prepyloric region The duodenal bulb was intact The stomach was moderately enlarged There was a 24-hour gastric residue The findings on postmortem examination of the obstruction of the pyloric outlet by the large tumor mass explained this abnormal retention

The mucosal appearance of the stomach in proliferative malignancy is well demonstrated by the following case

M V, male, aged 50 Six months be-



FIG 244 Note the irregular destruction of the mucosa produced by a carcinoma of the stomach.

fore hospitalization this patient began having dull pain in the epigastrium, at intervals lasting for 15 minutes but gradually becoming constant. During the preceding 6 weeks he had vomited daily, the vomitus being the food of the previous meal only. He had lost 15 pounds in the course of 1 year.

The report of the physical examination was as follows: "In the left upper quadrant is an ovoid mass, about $2\frac{1}{2}$ by $2\frac{3}{4}$ inches which is visible and palpable. The tumor starts in the epigastric region and runs along horizontally to the left and extends under the left costal margin. The tumor is firm in consistency, immobile on respiration, and is lobulated, a small nodular mass being present to the left of the main growth. The liver is palpable two fingers' breadth below the costal margin. Exploratory laparotomy revealed an inoperable malignancy of the stomach."

Roentgen examination (Fig 244) revealed a large translucent area occupying practically the entire pylorus, with an irregularly outlined margin of barium encircling this area. There was complete destruction of all normal mucosal markings in this region. In addition, there was an annular narrowing with irregularity of contour of the pars media proximal to the region just described.

The importance of compression technic

with spot films in demonstrating the mucosal structure in the diagnosis of a gastric malignant lesion is illustrated by the following case.

F L, female, aged 50. The patient gave a history of a few months of upper abdominal discomfort occurring about 2 to 3 hours after meals. She had lost about 3 pounds during this time. Physical examination was essentially negative. A subtotal gastrectomy was done.

The pathologic report was as follows: "On the anterior surface of the stomach, right next to the greater curvature and 1 cm from the proximal line of resection is a large tumor projecting into the lumen of the stomach. This is roughly oval in shape measuring 7 by 5 cm. It is moderately firm and extends through the musculature but not through the serosal surface. Elsewhere the mucosa of the stomach is not remarkable."

The microscopic examination revealed mucous cell carcinoma.

In the roentgen examination with the stomach completely distended with barium there was very little evidence of organic disease (Fig 245 A). It was only in the films taken with graded compression that the deformity produced by the tumor mass was clearly discernible (Fig 245 B). Note the fairly well circumscribed deformity pro-

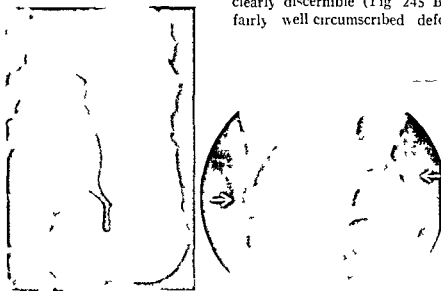


FIG 245 (A Left) Carcinoma of the pars media of the stomach. Because the stomach was distended with barium the deformity was not visible at this time. (B Right) Same patient illustrating the importance of graded compression in the demonstration of the deformity produced by the malignant lesion. Note the irregularly rounded translucent areas produced by the tumor.

duced by the tumor and the abnormal appearance of the mucosal relief within this region. One can readily understand from a comparison of these two roentgenograms why an intragastric tumor may be missed when the stomach is completely distended with barium unless compression is employed to bring into view the alterations of mucosal structure produced by the lesion.

That an essentially normal mucosal relief picture may be obtainable even when a tumor is present is to be explained by the fact that the mucosal folds that are visualized are those of the uninvolved wall of the stomach.

As noted in the operative report, the tumor was limited to the anterior wall, and the mucosa of the rest of the stomach was intact. The application of graded pressure, however, by forcing the anteriorly placed tumor against the posterior wall enabled us to visualize the abnormal pattern produced by the mass. This case proves the importance of using every method available in the roentgen demonstration of an organic lesion: (1) study of the gross contour, (2) study of the mucosal relief, and (3) use of the compression technic.

How completely malignant changes in the mucosal structure may be obscured when the stomach is completely distended with barium is illustrated by the next case.

C A, aged 50. This patient gave a history of several months of vague upper abdominal discomfort. There was only moderate weight loss. Physical examination was essentially negative. At operation, there was definite thickening of the distal portion of the stomach. A small neighboring lymph node was felt to be somewhat firm. A subtotal gastrectomy was done. The pathologic diagnosis was ulcerated infiltrated colloid adenocarcinoma of the stomach with early involvement of a lymph node.

Roentgen examination with the stomach filled with barium (Fig 246 A) showed no definite evidence of new growth. The mucosal study (Fig 246 B) showed a number of irregularly rounded translucent areas occupying the distal portion of the stomach. Based on this appearance a diagnosis of malignancy of the stomach was made.

An unusual mucosal appearance produced by an adenocarcinoma of the stomach is illustrated by the following case.

D H, female, aged 36. The patient gave a history of vague digestive complaints over a period of about 4 years. She stated that she had been treated for ulcer during that time.

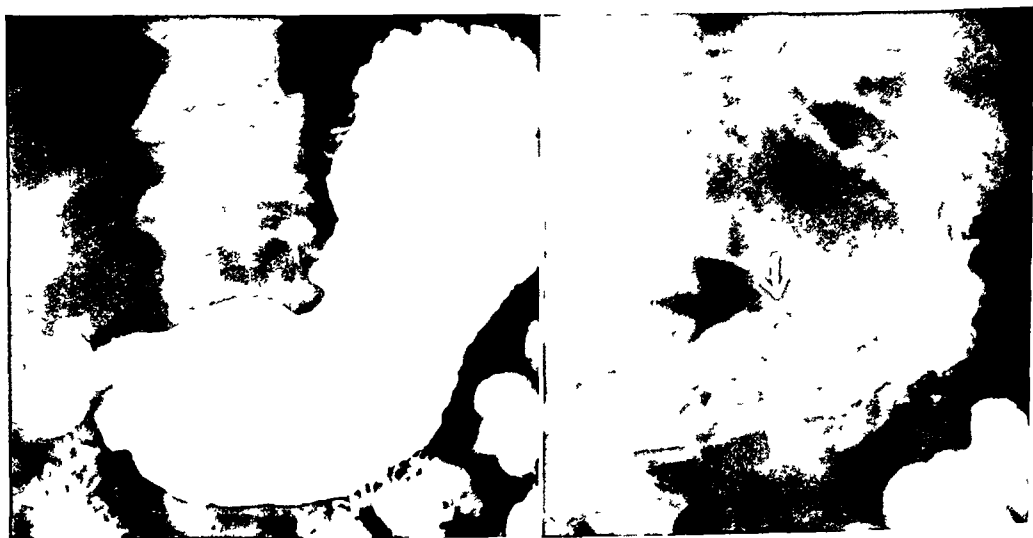


FIG 246 (A, *Left*) Carcinoma of the stomach. The deformity is obscured by barium. (B, *Right*) Same patient. Mucosal relief appearance, showing destruction of the mucosa produced by the new growth.



FIG 247 (A *Left*) Changes in the mucosa produced by carcinoma simulating the appearance of prominent mucosal folds (B, *Center*) Same patient Note in particular the fingerlike areas projecting into the air bubble (C *Right*) Appearance of resected specimen Note the prominent tortuous folds of the stomach simulating the appearance of cerebral gyri

She had also complained of an occasional nonproductive cough and of pain across the lower abdomen and back.

Roentgenographic examination of the chest revealed a mass in the chest the exact nature of which was not determined. Gynecologic examination revealed multiple fibroids, a rectocele and lacerated perineum. Abdominal examination did not disclose any masses or tenderness.

Abdominal operation undertaken because of the roentgen findings revealed a markedly enlarged stomach with walls almost $\frac{3}{4}$ of an inch thick. This involvement extended from the duodenum to the esophagus. There were no obvious nodes present. The rest of the alimentary tract was normal. The liver and the spleen were normal.

Pathologic examination revealed the following. On opening the stomach the rugae were found to be unusually large and numerous with their mammillae measuring from 4 to 10 mm in size. The upper three fourths of the stomach showed this change. The pyloric fourth had only flattened rugae. The mucous membrane was whitish gray covered with reddish mucoid material. In the upper three fourths of the stomach the wall measured up to 1.4 cm in thickness. This was due to thickening of the mucosa and edema of the submucosa. There were two areas of ulceration near the fundus of the stomach. One measured 1 cm in diameter was irregular in outline and contained in its crater an 8 mm solid rounded whitish yellow mass extending from the level of the surface of the mucosa through the mucosa and submucosa to the muscularis. The muscularis appeared normal.

The other ulceration measured about 5 cm in diameter, with irregular border and crater and contained blood stained material. Sections were taken through the ulcer, the fundal border of the stomach and the pyloric region.

The report of the microscopic examination was: "Sections reveal the gastric mucosa thickened and the submucosa edematous. In certain areas of the sections atypical epithelial elements are seen growing through the basal portion of the mucosa in the form of irregular cords. The section through the ulcer reveals a rounded lobulated mass of tumor tissue composed of double rows of hyperchromatic small cells with their nuclei arranged along the bases of the cells at the outer margins of the pseudotubule formations. These formations branch irregularly and the tumor is seen invading the submucous tissue. The tumor tissue and the submucosa in the base of the ulcer are edematous and invaded by many polyps. Section of omental lymph node reveals the presence of adenocarcinomatous tumor tissue."

The diagnosis was carcinoma of the stomach with metastases in a lymph node.

This is a very unusual carcinoma of the stomach. The histologic picture suggests the possibility of a diffuse carcinomatous involvement of the mucosa. The cell type is such that the tumor may have arisen from the acid cells of the mucosa.

Roentgen examination (Fig 247 A and B) revealed unusual prominence of the mucosal folds throughout the entire stomach. These changes were particularly prominent in the proximal portion of the stomach and gave the



FIG 248 (A, *Left*) Adenocarcinoma of the pars cardia invading the lumen from the greater curvature side (B, *Center*) Appearance of the resected adenocarcinoma (C, *Right*) Appearance of the well-functioning postoperative esophagogastrostomy

impression of fingerlike masses extending well into the gastric air bubble. They were abnormally wide, of irregular outline, rigid and fixed. The entire picture was that of an abnormal mucosa with extremely prominent, wide and rigid folds involving mainly the proximal portion of the stomach and a more polypoid type of involvement of the distal area. Although it has been my experience

that it is impossible on the basis of the radiologic evidence to differentiate a lymphosarcoma from a carcinoma, it was suggested that a lymphosarcoma might explain the roentgen findings. This assumption was based particularly on the roentgen appearance of the pars cardia. It was not believed that the broad, fingerlike masses in this area could be explained on the basis of a chronic gastritis.

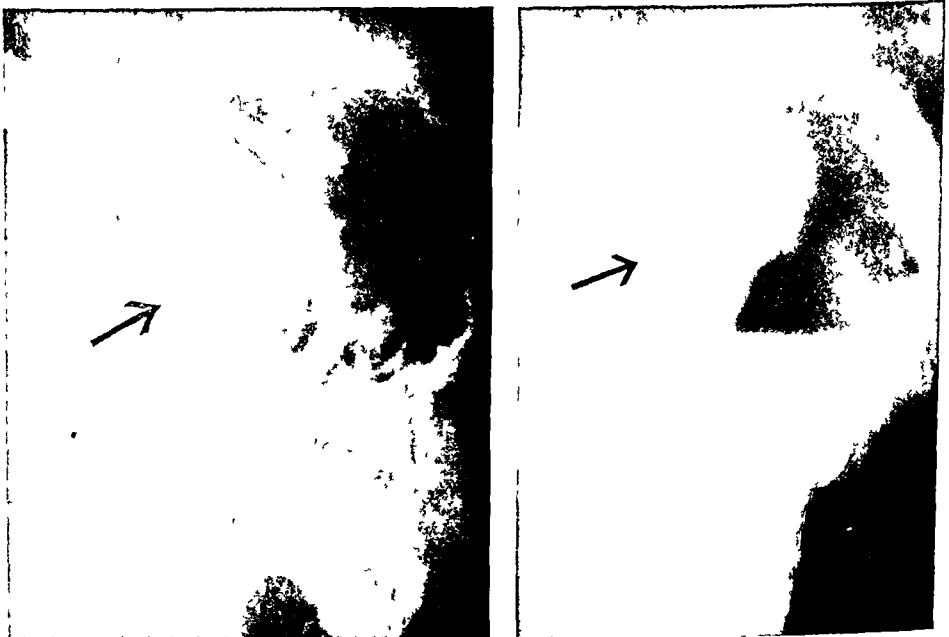


FIG 249 (*Left*) Carcinoma of the cardia invading the gastric air bubble
FIG 250 (*Right*) Malignant tumor invading the gastric air bubble

As noted on pathologic examination the lesion proved to be an unusual type of adenocarcinoma.

The gross appearance of the resected specimen (Fig 247 C) shows the unusually prominent gastric folds looking like cerebral gyri.

Carcinoma of the cardiac portion of the stomach may deform the lesser or the greater curvature or show its presence by invasion of the air bubble. The following case illustrates a carcinoma of the cardiac portion of the stomach deforming the greater curvature.

J N male, aged 43, gave a 10 month history of recurrent episodes of epigastric pain relieved by food. He lost 17 pounds. On one occasion he had a tarry stool. Physical examination of the abdomen was essentially negative. Roentgenographic examination (Fig 248 A) revealed a large irregular defect of the greater curvature of the stomach pars cardiaca. Through a transthoracic abdominal approach a large carcinoma of the fundus of the stomach was found adherent to the diaphragm to the left of the esophagus. The tip of the left lobe of the liver was fused to the cardiac end of the stomach. The carcinoma extended into the hilar region of the spleen and the region of the tail of the pancreas. The tissue contained some lymph nodes but no other lymph nodes were found outside the dissected area.

The proximal portion of the stomach, the spleen, the tail of the pancreas, the tip of the left lobe of the liver and the lower end of the esophagus were resected and an esophago-gastrostomy was done.

The diagnosis on pathologic examination was adenocarcinoma of the stomach with extension to the peritoneum and the mesentery. Sections through 18 additional lymph nodes failed to disclose any evidence of metastatic tumor.

Figure 248 B shows the appearance of the resected carcinoma of the stomach. Figure 248 C shows the postoperative roentgen appearance of the well functioning esophago-gastrostomy.

The reason for the deformity of the greater curvature of the cardiac portion of the stomach may be understood by comparison with the appearance of the resected tumor.

Of considerable interest is the invasion and distortion of the gastric air bubble by

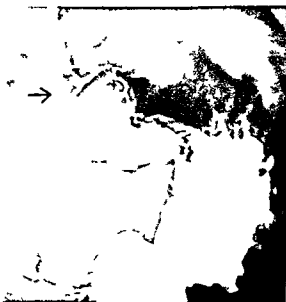


FIG 251 Carcinoma of the pars cardiaca. Note the deflection of the barium produced by the tumor.

a new growth. In some cases, this can be recognized by direct fluoroscopic inspection without the administration of barium. A thin layer of barium sticking to the tumor may further enhance its visibility in the roentgenogram. The following case illustrates this.

E N female, aged 50 years. Nine months before hospitalization this patient developed pain to the left of the epigastrium. This pain occurred immediately after eating. At the time of admission the pain had increased in severity and was relieved by forced vomiting. There was no blood in the stool. She had lost 25 pounds in 8 weeks. Examination of the abdomen revealed a resistance in the midepigastrium. At operation there was a carcinoma high up on the lesser curvature of the stomach.

Roentgen examination (Fig 249) revealed a tumefaction at the cardiac end of the stomach invading and deforming the air bubble. There was secondary obstruction at the cardiac end of the esophagus so that the entire esophagus was distended with barium.

Figure 250 is a malignant tumor showing as a soft tissue mass invading the gastric air bubble at the lesser curvature. The tumor shows with particular clearness

tumor was resected, including the spleen and the distal end of the esophagus, and an esophagogastrostomy was done

Pathologic examination Adenocarcinoma of the stomach with extension to the esophagus, chronic lymphadenitis of gastric lymph nodes The spleen revealed no pathology

The roentgenographic examination is of considerable interest from the standpoint of the technic of the examination (Fig 254 A and B) Figure 254 A represents the appearance on examination of the patient in the prone position so that the cardiac portion of the stomach is distended with barium There is no evidence of tumefaction Figure 254 B shows the appearance when the examination was made with the patient in the erect position The gastric air bubble is well visualized The encroaching tumor is noted invading and deforming that portion of the air bubble near the lesser curvature In addition there is marked irregularity in the configuration of this area as outlined by the barium Obvious as is the evidence of an invading tumor in Figure 254 B, its presence was completely obscured because of the overdistention of this region with barium in Figure 254 A

An isolated, well-circumscribed malignancy may closely simulate roentgenologically the appearance of a benign tumor

A Z, male, aged 70. During the 6 weeks before his admission to the hospital, the patient had complained of weakness and occasional dizziness The stools were tarry in color at the onset and his local doctor told him that he was anemic He had lost 10 pounds since the onset He also had complained of dyspnea and a generalized edema, which disappeared with bed rest and medication At no time did he have any abdominal pain or vomiting

Physical examination was essentially negative, except for marked anemia and a moderate edema of the ankles and dorsa of the feet Abdominal examination was essentially negative

Gastroscopy revealed an elliptical tumor mass in the pars cardia on the anterior wall near the lesser curvature attached by a broad base It measured about $2\frac{1}{2}$ cm in its longest diameter The margins of the neoplasm were well defined The surface was grayish white in color and lobulated in design In the center was a deep-red hemorrhagic area The impression of the gastroscopist was neoplasm of the stomach, which might be a leiomyoma or possibly a leiomyosarcoma with central hemorrhagic ulceration

An almost total gastrectomy was done

The macroscopic examination revealed a firm movable mass in the cardia, fungating



FIG 254 Carcinoma of the stomach in the prone position (A) and in the erect position (B) at 90°. Note the irregularity of contour of the cardiac portion of the stomach in the erect position (B), which is completely obscured by the overdistention of the cardiac portion of the stomach in the prone position (A).

diac portion of the stomach is no evidence of tumor on (B, Right) examination. The gastric air bubble is well visualized, and the irregularity of contour of the cardiac portion of the stomach is completely obscured by the overdistention of this region with barium in Figure 254 A.

(A, Left) Examination of the cardiac portion of the stomach in the erect position (B) shows the tumor, as well as the irregularity of contour of the cardiac portion of the stomach.

and cauliflowerlike in appearance, roughly ovoid, being 4 cm and 6 cm between opposite poles and about $3\frac{1}{2}$ cm thick, attached to the lesser curvature by a fairly broad base and almost occluding the lumen of the stomach.

Microscopic examination revealed papillary adenocarcinoma of the stomach.

Roentgen examination (Fig 255 A) revealed a fairly large, rounded translucent area high up in the pars cardia and intraluminal placed. While it was quite obvious that we were dealing with a tumor of the pars cardia, the radiologic appearance suggested that the tumor might be benign. It is interesting to note that the gastroscopist considered that the lesion might be a leiomyoma or possibly a leiomyosarcoma. The photograph of the specimen (Fig 255 B) shows a well circumscribed polypoid mass corresponding quite closely to the translucent area noted in the roentgenogram. It was the pathologic examination which established the nature of the lesion.

The simulation in the roentgenogram of a benign tumor of the stomach by a carcinoma is further illustrated by the following case.

E. B., male, aged 75. The patient had complained of increasing weakness during the year preceding his hospitalization and had lost 25 pounds. He had no digestive disturbances. On one occasion he had vomited coffee grounds material, positive for blood.

Physical examination of the abdomen was essentially negative. He was markedly anemic, and there was evidence of Paget's disease. Gastric analysis showed an absence of free hydrochloric acid. The stools were persistently positive for blood.

The operative findings were reported on as follows: In the pars media of the stomach there was a large polypoid growth with a sessile base protruding into the lumen of the stomach. It was very hard, nodular in outline, all layers of the stomach and extending into a cauliflowerlike excrescence on the serosal surface of the posterior wall of the stomach. It gave the impression of being a malignant degeneration of a polyp. No metastases were made out anywhere.

A partial gastrectomy was done.



FIG 255 (A Left) Adenocarcinoma of the stomach. (B Right) Same patient. Note the well circumscribed tumor corresponding to the translucent area in the roentgenogram.

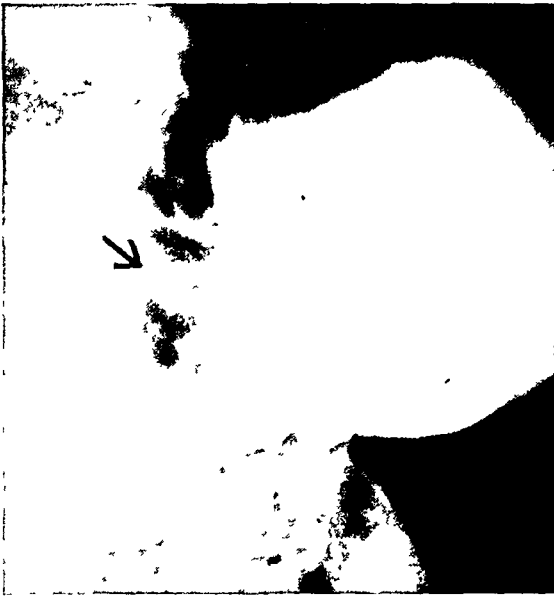


FIG 256 Malignant polyp of the stomach

The pathologic report was adenocarcinoma of the stomach of the mucoid type with metastases

Roentgen examination (Fig 256) revealed a fairly well-rounded, intraluminally placed translucent area within the confines of the pars media, having some of the characteristics of a benign lesion. While there was obvious evidence of a tumor, it was suggested on ra-

diologic grounds that the tumor might be benign. As noted on pathologic examination, the lesion proved to be malignant. This case is an indication of the difficulty that may be encountered in clearly differentiating on the basis of the roentgen evidence between a benign and a malignant tumor and the advisability of partial gastrectomy, particularly if the tumor has a sessile base.

The occasional difficulty of differentiating on the basis of the roentgenologic findings between a benign and a malignant lesion of the stomach is also illustrated by the following case.

J G, male, aged 68. During the 4 months preceding his hospitalization, the patient had complained of epigastric pain. He was unable to give a clear history because, as he stated, "My mind is all mixed up." Physical examination disclosed a hard mass in the left upper quadrant about 5 cm in diameter. There was a severe anemia. Free acid was present in the gastric contents only after the injection of histamine. On gastroscopy a circumscribed mass was noted projecting from the lesser curvature, pars media, into the lumen of the stomach. The surface was covered with normal-looking mucosa. No ulcerations were seen. The rest of the gastric mucosa appeared normal. It was the impression of



FIG 257 (A, *Left*) Malignant tumor of the stomach, simulating the appearance of a benign lesion. (B, *Right*) Same patient. Appearance of the resected specimen.

the gastroscopist that the findings were due to a benign polyp.

A subtotal gastrectomy was done. Examination of the resected specimen showed a tumor mass in the distal portion of the pars media about 5 cm in diameter. The tumor was well demarcated. There were two nodes on the lesser curvature. The liver was free of invasion.

Pathologic examination revealed an irregular polypoid mass 5 cm in diameter over which the mucosa seemed to be intact. The growth infiltrated the wall and protruded onto the external surface at three points where however it was still covered by serosa. The cut section revealed firm white tissue.

The microscopic diagnosis was adenocarcinoma of the stomach.

Roentgen examination (Fig 257 A) revealed a rounded, well circumscribed, intraluminal translucent area in the pars media near the lesser curvature. Because of the roentgenologic appearance it was believed that the lesion was a polyp, probably benign. As noted on pathologic examination, the tumor was an adenocarcinoma. Figure 257 B shows the gross appearance of the tumor.

The difficulties involved in the differentiation of a benign from a malignant polyp of the stomach and the practical implications which this involves are further illustrated in this case.

G. P., female, aged 71. The patient gave a 2 month history of headache, dizziness, and periumbilical pain usually occurring about 1/2 hour after meals. There was occasional vomiting. She had lost some weight, but the exact amount could not be determined. Physical examination revealed a globular mass in the left upper quadrant of the abdomen. Roentgenographic examination revealed evidence of a fairly large irregularly outlined tumor within the confines of the pars media of the stomach (Fig 258). Because of the relative size and irregular configuration of the intraluminal translucency, I was of the opinion that the tumor was probably malignant.

Operation revealed a pedunculated tumor attached to the posterior wall of the stomach, closer to the lesser than to the greater curvature, about 6 cms proximal to the pylorus.

The tumor appeared to be a nonmalignant variety with little evidence of infiltration of the surrounding stomach wall. It was free upon its pedicle and apparently dropped down into the pylorus acting as a ball valve. The



FIG 258 Note the irregularly outlined translucent area produced by a malignant polyp of the stomach.

tumor was irregular and nodular and measured about 10 cms at its greatest diameter. On the lesser curvature of the stomach there was a large node measuring about 3 cm in diameter with some evidence of extension along the lymphatics up toward the esophagus.

Because of the general poor condition of the patient it was believed that she would not tolerate a subtotal gastrectomy. A local removal of the tumor was done, including a small portion of the surrounding normal mucosa. However, there was some infiltration of the muscular wall of the stomach in the area of the resection.

Pathologic diagnosis: Adenocarcinoma of the stomach.

Therefore, in spite of the fact that the polyp was attached by a pedicle and the surgeon considered the tumor to be benign, it proved to be malignant. A subtotal gastrectomy would have been the operation of choice provided that the condition of the patient had justified this more radical procedure.

The grave danger of assuming that a polyp is benign from its gross appearance, even when the surgeon is able to shell it out of its bed, is illustrated by the following case. L. P., female, aged 60. I believe also that it is the type of evidence which tends to favor subtotal gastrectomy rather than localized removal of the gastric tumor, particularly in the absence of a pedicle, even though the tumor may appear to be quite

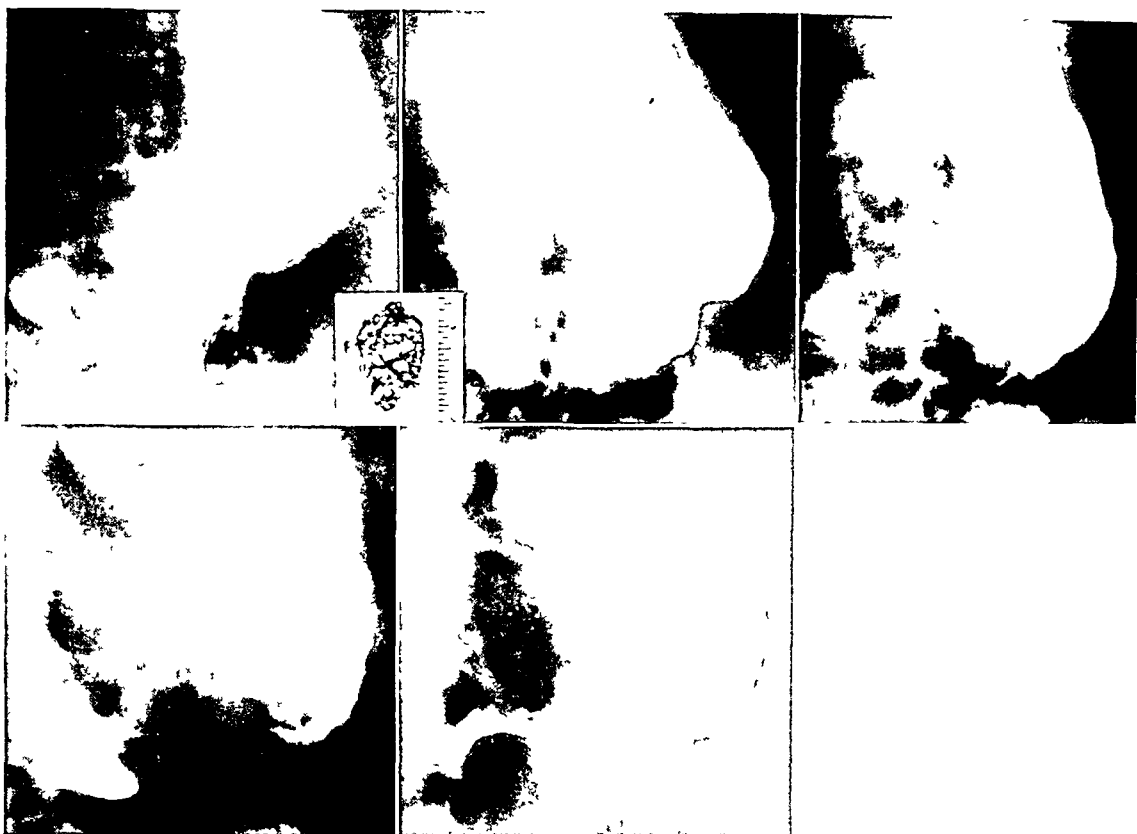


FIG 259 Malignant polyp of the stomach (A, *Top, left*) Note the rounded intraluminal translucencies in the prepyloric segment of the stomach at the time of the original examination. The evidence was considered as probably due to a benign tumor (B, *Top, inset*) Appearance of the resected tumor which was shelled out by blunt dissection (C, *Top, center*) Appearance 11 months after the first operation. Note the spread of the deformity (D, *Top, right*) Four months after (C) Note the exaggeration of the deformity of the pylorus. Exploration at this time revealed an inoperable carcinoma with metastases (E, *Bottom, left*) Appearance 4 months later. Note the marked exaggeration of the deformity of the stomach (F, *Bottom, right*) Appearance 10 months after (E) Note the further spread of the deformity. All of these figures enable us to trace the spreading deformity of an originally localized encapsulated tumor.

benign to the surgeon and may possess roentgen characteristics substantiating that assumption.

The patient was admitted to the hospital because of progressive dyspnea and ankle edema. She had lost weight and was markedly anemic. Physical examination of the abdomen was negative. Gastric analysis showed no free hydrochloric acid. The Wassermann was 4 plus.

Roentgenographic examination at that time (Fig 259 A) showed small rounded intraluminally placed translucent areas in the prepyloric region, and a diagnosis of new growth was made. I thought that the tumor might well be benign. She left the hospital without

being operated and was followed in the outpatient department. About 6 months later she was sent into the hospital for operation because of the roentgenographic findings. At operation a single isolated, flat apparently benign tumor was found on the posterior wall of the pyloric antrum. The tumor measured about 4 x 3 cm and, according to the surgeon, did not extend below the submucosa. He believed that the tumor was benign. On entering the stomach, the tumor was secured, the mucosa was incised, and by blunt dissection along the submucosa the mass was freed from its attachments. Figure 259 B shows the appearance of the excised tumor.

In spite of the suggestive roentgenographic

appearance and the anatomic characteristics of the lesion, the histologic report was carcinoma. After discharge from the hospital she remained under continued observation in the outpatient department and repeated roentgenographic examinations were made. Whatever doubt might have been entertained regarding the malignant nature of the lesion was dissipated by the findings on roentgenographic examination which showed an increase in the degree of deformity (Figs 259 C and D). Figure 259 C shows the appearance 11 months after Figure 259 A and 4 months after the first operation. Figure 259 D represents the appearance about 4 months after Figure 259 C, or 1 year and 3 months after Figure 259 A. At the time of this second examination (Fig 259 C) there was definite persistent irregularity of the distal segment of the greater curvature of the stomach as well as a small area of irregularity on the lesser curvature border. There was no doubt in my mind that we were witnessing the spread of a malignant lesion. However, the surgeon was loath to reoperate believing that the distortion in the roentgenogram might be accounted for by the operative procedure. Whatever lingering doubt might have been entertained as to the nature of the lesion was completely dispelled by the roentgenographic appearance noted in Figure 259 D 4 months later. Note the marked spread of the deformity of the distal portion of the stomach.

Reoperation 10 months later disclosed the following:

1 The liver was markedly enlarged. There were several large irregular firm lymph nodes. In the greater omentum there were several hard irregular masses.

2 Within the stomach was a large fungating friable tumor mass, extending into the lumen of the pars pylorus and the pars media. An incision was made into the stomach and a piece of the tumor mass was excised. A hard nodule in the greater omentum was also removed by dissection.

Pathologic diagnosis: Adenocarcinoma of the stomach with metastatic carcinoma.

From then on she went progressively downhill and died 13 months after the second operation. The continued progress of the deformity is to be traced in the succeeding roentgenograms.

Figure 259 E shows the appearance 4 months after Figure 259 D and 3 months after the second operation. Figure 259 F represents the appearance 10 months after Figure 259 E. Note the progressive spread of the malignant lesion. By a study of these different figures one may literally see the unfolding and the gradual spread of a malignant lesion from a small localized focus to invasion of most of the stomach.

In retrospect it appears quite obvious that a subtotal gastrectomy should have been done instead of a local excision of the tumor. It is

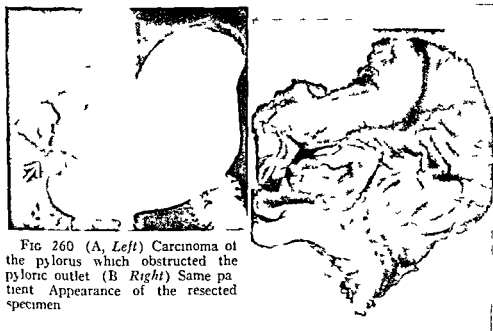


FIG 260 (A, Left) Carcinoma of the pylorus which obstructed the pyloric outlet (B Right) Same patient. Appearance of the resected specimen.

further evidence of the danger of assuming that a gastric tumor is benign, not only on the basis of the roentgenographic evidence but also on the gross characteristics of the lesion at the time of operation

A malignant lesion of the pylorus may produce marked obstruction

C M, female, aged 40 The patient gave a 3-month history of epigastric distress occurring about 1 hour after meals and lasting about $\frac{1}{2}$ hour She had lost 13 pounds in that period Physical examination of the abdomen was essentially negative Operation revealed a tumor of the pars pylorica, for which a subtotal gastrectomy was done (Fig 260 B) The pathologic diagnosis was cylindrical-cell carcinoma of the stomach

Roentgen examination (Fig 260 A) revealed marked narrowing of the pars pylorica The lesser curvature in the distal portion of the stomach was smooth, rigid and devoid of peristalsis The greater curvature in the constricted zone was very irregular and showed an abrupt change from the contour in the rest of the stomach There was considerable gastric enlargement and retention at 48 hours The preoperative diagnosis was carcinoma of the pylorus with obstruction

The delimitation of a scirrhous infiltra-

tive lesion may to some extent be demonstrated by a study of the mucosal relief This is well shown by the next case

M A, male, aged 56 This patient's main complaint was of lower abdominal discomfort occurring just before eating, and of a feeling of gaseous distention over a period of 6 months He had lost no weight Physical examination of the abdomen was essentially negative

The operative findings were an infiltrating lesion involving the anterior and posterior surfaces of the proximal two thirds of the stomach, somewhat more extensive on the posterior surface where it was adherent to the pancreas, these adhesions seemed to be inflammatory rather than neoplastic The growth had the appearance and felt like a linitis plastica accompanied by a considerable amount of inflammatory reaction There were no metastases in the nodes or in the liver, but there was an area of direct extension into the gastroduodenal omentum for a distance of over 1 inch from the greater curvature An almost total gastrectomy was done

The report of the macroscopic examination of the resected specimen was as follows "There is a punched-out ulcer, $\frac{1}{2}$ inch in diameter, along the greater curvature midway



FIG 261 Mucosal appearance in scirrhous infiltration of the stomach Note the termination of the normal mucosal folds in the region of the new growth The lower arrow points to the ulcer within the malignant lesion.

between the sites of resection. The anterior and the posterior surfaces show an area of induration about 2 sq in each. Microscopic examination: Infiltrating carcinoma of the stomach. The proximal end of the resected specimen shows no involvement by tumor. Examination of a lymph node shows lymphocytic extension of gastric carcinoma."

Roentgen examination (Fig 261) revealed an infiltrative scirrhus type of malignant new growth. At the greater curvature in the involved area was the projecting niche representing the ulceration found in the resected specimen. Particularly instructive is the study of the mucosal folds. These run in a normal parallel manner throughout the pylorus. At its junction with the pars media there is an abrupt termination of these folds, the mucosal folds disappearing because of their destruction by the new growth. The greater curvature pars media is stiff and superficially irregular with involvement of the pars cardia.

Rarely there may be a metastatic implantation of the stomach from a primary malignant lesion originating in a distant focus.

C. R. male, aged 63. The patient stated that he had been well until 2 weeks before his admission to the hospital when he noticed a gradually progressive weakness in his arms and legs and on occasion a vague epigastric soreness. There had been no vomiting and no blood in the stool.

Physical examination disclosed a cadaverous looking elderly white male in no acute distress. On abdominal examination the liver edge was felt two fingers breadth below the costal margin. The prostate was described clinically as being essentially normal.

The autopsy report was as follows: On the anterior aspect of the wall of the stomach near the cardia are two small moderately firm nodules about 1 cm in diameter. The mucosa over these areas is smooth. On section these nodules are yellowish in color and homogeneous. The prostate is about twice its normal size. It is firm in consistency and its surface is studded with irregular hard nodules about 1 cm in diameter. The microscopic examination revealed adenocarcinoma of the prostate with metastases to lymph nodes, liver, spleen, ribs, vertebrae, adrenal glands and stomach.

Roentgen examination of the stomach (Fig 262) revealed a small rounded translucent area in the pars cardia which it was thought might represent a polyp. Further studies as

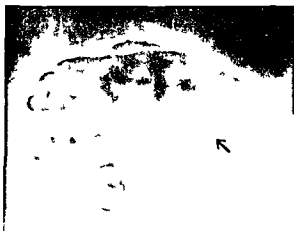


FIG 262 Metastatic tumor of the stomach from a carcinoma of the prostate. Note the rounded translucent area produced by the tumor.

well as gastroscopy were not carried out because of the patient's physical condition. As noted in the autopsy examination, this polypoid mass was the result of metastatic invasion of the stomach from a primary carcinoma of the prostate.

Marked deformity of the stomach because of secondary invasion occurred in the next case.

E. K. male, aged 75. The patient gave a 1 month history of abdominal pain of increasing severity. He had vomited on three occasions but no blood was present. He had had no bowel movement in the preceding 4 days. He also complained of dyspnea and of attacks of precordial pain during the past few years. He had lost 18 pounds in the preceding 2 weeks. Two years previously he had had an attack of epigastric pain and was hospitalized for 2 months. On physical examination the abdomen was found to be distended and tender all over. Examination of the chest was negative.

Gastric analysis showed an absence of free hydrochloric acid. At operation the stomach was reported as being perfectly normal throughout its entire extent until the pylorus was reached. A stony hard mass was then encountered measuring about 8 x 10 x 8 cms in size. This mass extended from the region of the second and third portions of the duodenum laterally to the right lumbar gutter and invaded and extensively involved the transverse mesocolon. It had also invaded the

omentum Again it was stated that the stomach was of normal size and seemed to show no evidence of obstruction at the duodenum A diagnosis of retroperitoneal tumor of undetermined origin was made

At autopsy it was reported that examination of the lungs revealed a carcinoma of the bronchus in the right lower lobe There was secondary carcinoma in the lungs, stomach, lesser omentum, liver, heart, seventh right rib and transverse colon The findings in the examination of the stomach are of considerable interest "As the stomach is opened, it is apparent that the wall of the distal half, to, but not including, the pylorus is infiltrated by a firm tumor There is no obvious erosion of mucosa The lesser curvature seems to be the point of origin, but the growth extends on to the greater curvature . The wall of the stomach is greatly thickened at the site of the tumor, and section through it reveals dense, homogeneous, gray tumor tissue which had apparently replaced the muscularis and completely obliterated the various layers The tumor tissue involves the serosa, not only at its point of fusion to the liver but over a wide area The tumor tissue has extended into the adipose tissue of the lesser omentum so that the latter is thickened and very solid "

Roentgen examination showed an extensive narrowing with irregularity of contour involving primarily the distal two thirds of the stomach (Fig 263) The preoperative report was carcinoma of the stomach As noted at autopsy, the infiltration of the stomach by tumor tissue was secondary to a primary bronchogenic carcinoma

A malignant lesion of the stomach may ulcerate into the colon, causing a gastrocolic fistula or, as in the case to be described, the fistula may be localized

J E, female, aged 67 During the 4 days preceding her hospitalization the patient had complained of generalized, crampy abdominal pain, which finally localized to the right upper quadrant She had been vomiting occasionally during the preceding 2 weeks After taking an enema she noted that the return was tarry in color Physical examination revealed a tender mass on the right side of the abdomen

Laparotomy disclosed a carcinoma at the greater curvature of the pylorus which had perforated with the production of a fistulous tract extending downward in the direction of the right lower quadrant A specimen was removed from the tumor for biopsy No further surgery was attempted because of the patient's condition

Microscopic examination showed the lesion to be adenocarcinoma

Roentgen examination (Fig 264) revealed considerable narrowing at the pyloroduodenal region, with secondary gastric enlargement At no time was the duodenal bulb visualized A fistulous tract was noted, extending from this area downward toward the right lower quadrant It was believed preoperatively that the fistula originated from the duodenum and not from the stomach itself No lesion of the colon was disclosed by barium-enema examination



FIG 263 (*Left*) Metastatic infiltration of the stomach from a primary bronchogenic carcinoma



FIG 264 (*Right*) Carcinoma of the pylorus, with fistulous tract

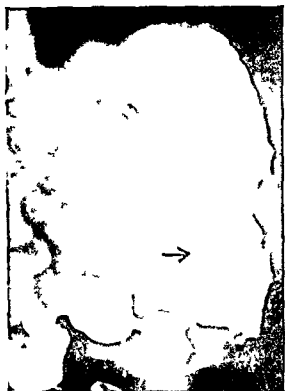


Fig 265 (Left) Reticulum cell sarcoma of the stomach

Fig 266 (Right) Lymphosarcoma of the stomach

As noted at operation the cause of the fistulous tract was a carcinoma of the greater curvature of the pars pylorica which had perforated and become walled off

Sarcoma of the stomach is roentgenologically indistinguishable from carcinoma

B S, male, aged 63 The patient gave a 10 month history of recurrent attacks of intense epigastric pain which kept him awake at night At times, the pain radiated to the back There was occasional vomiting

Examination at operation revealed a malignant lesion involving the entire pars media with invasion of the serosa and with metastases to the omentum On the lesser curvature side it was found to be adherent to the left lobe of the liver It was obviously inoperable The biopsy specimens revealed reticulum cell sarcoma

Roentgen examination (Fig 265) revealed a diffuse, irregular narrowing of almost the entire stomach characteristic of malignant invasion There was nothing in the roentgen appearance to indicate that the malignant process was a reticulum cell sarcoma The deformity was identical with that produced by an adenocarcinoma

In the following case the deformity pro-

duced by a lympho sarcoma mimicked that of a sarcoma

I C, male, aged 57 The patient gave a 4 month history of dull epigastric pain associated with pain in the chest He noticed black stools on occasion He had lost 36 pounds during the 7 months preceding hospitalization

The physical examination showed there was a mass in the epigastrium about 3 inches in diameter firm, nodular and relatively fixed There was a right rectus scar as the result of a cholecystectomy 5 years previously Gastric analysis showed no free hydrochloric acid The stool was positive for blood

At operation the surgeon reported an inoperable carcinoma of the stomach with metastases A node at the greater curvature was removed for histologic examination The microscopic examination revealed lympho sarcoma

Roentgen examination (Fig 266) revealed marked narrowing of the pars media with irregularity of contour The findings were reported as indicating the presence of a carcinoma Only the pathologic examination indicated that the lesion was a lymphosarcoma There was nothing in the roentgen appearance which might have aroused a suspicion that

the deformity was the result of a lymphosarcoma rather than a carcinoma

The impossibility of differentiation between lymphosarcoma and carcinoma on the basis of the roentgen findings is further illustrated by the next case.

B B female, aged 65 This patient gave a 2-month history of gnawing pain in the upper abdomen which was not related to food There was no vomiting She had suffered only a moderate weight loss of 4 pounds in the 2 months preceding her hospitalization She had noticed blood in the stool

Operation revealed an apparently inoperable malignancy of the stomach A biopsy was done The pathologic report reads as follows

Gross specimen consists of three small pieces of the wall of the stomach, the largest measuring 3 cm by 2 cm by 1 cm The inner surface shows an attached mass, which is softer than the stomach wall proper and friable The stomach itself shows rather distinct markings with whitish-yellow, opaque strands throughout Microscopic Section through the stomach shows necrotic mucosa and abundant infiltration of all the coats, especially the muscular coat, with immature lymphoid tissue showing frequent mitotic figures and a large amount of reticulum cells definitely varying in size and shape The cells of the lymphoid tissue appear rather small Diagnosis Lymphosarcoma of the stomach"

Radiologically, the appearance is like that of a malignant infiltration of the stomach due to carcinoma There are no distinguishable roentgen features in this case Figure 267 shows the diffuse irregularity of contour of the stomach

A number of interesting features, pathologic and roentgenographic, are illustrated by the following case

F C, Chinese laundryman, aged 31 During the 4 weeks preceding his hospitalization the patient had noticed a progressive enlargement of the cervical, supraclavicular and axillary nodes There was marked weakness and a weight loss of 11 pounds There were no gastro-intestinal symptoms at the time of his admission to the hospital but while under observation he complained of considerable epigastric pain, abdominal distention and vomiting Physical examination revealed firm, nontender, pea-sized nodes in both the anterior and the posterior cervical regions Over both the mastoid and the parotid areas there were firm, rubber-hard movable nodes approximately 2 cm in diameter In the right supraclavicular area there was a large discrete node 3 cm by 2.5 cm There were pea-sized nodes in both infraclavicular areas, irregular masses of nodes in both axillae and chains of small nodes beneath the skin of both arms In the left femoral triangle there was a node 2 cm by 2 cm Neither the liver nor the

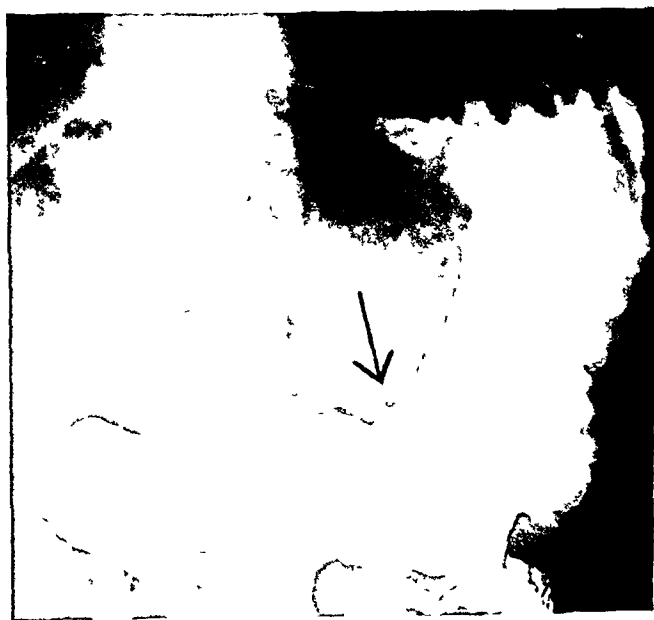


FIG 267 Lymphosarcoma of the stomach

spleen was palpable, but an indefinite mass was felt in the right upper quadrant.

There were no metastatic deposits in the bony framework of the chest or in the lungs on roentgen examination nor was there any pathologic condition of the lumbar spine or pelvis.

Microscopic examination of a node was reported by Dr Douglas Symmers as follows:

"Examination of slide shows the presence of what I believe to be lymphosarcoma, the cells of which are of the type of large rather than small lymphocytes. There is nothing to indicate the histology of Hodgkin's disease. The distribution of the enlarged lymph nodes is quite in keeping with the diagnosis of lymphosarcoma, but the presence of vast numbers of eosinophiles in the microscopic preparation taken into consideration with the enlarged lymph nodes suggests the possibility of eosinophilic leukemia. The latter disease not infrequently, however, is associated with enlargement of the spleen. Absence of enlargement on the other hand does not eliminate the diagnosis of eosinophilic leukemia."

As a result of x-ray therapy most of the nodular enlargement subsided with the exception of the right supraclavicular node.

Roentgen examination (Fig 268 A) with the stomach filled with barium showed no

evidence of organic abnormality. The mucosal relief study (Fig 268 B) showed marked deformity of the stomach involving primarily the distal half with destruction of most of the folds in this region. The process appeared to stop abruptly at the base of the duodenal bulb. The folds in the proximal portion of the stomach were retained although they were quite prominent. In both roentgenograms there was considerable narrowing of the jejunum just distal to the duodenojejunal junction. The folds, however, appeared to be retained. The diagnosis was malignant infiltration involving primarily the distal half of the stomach. The narrowing of the jejunum was considered as probably due to metastatic implantation. In view of the clinical findings and the biopsy report one may reasonably assume that the deformity of the stomach was the result of a lymphosarcoma or possibly eosinophilic leukemia. The narrowing of the jejunum may well be explained on the basis of associated involvement by the same pathologic process.

Lymphosarcoma may show roentgen evidence simulating the appearance of a hypertrophic mucosal fold.

S. A., female, aged 55. In the year preceding her hospitalization this patient had lost



FIG 268 (A Left) Lymphosarcoma of the stomach. The deformity is obscured at this time. (B Right) Same patient. Mucosal relief study showing the deformity produced by the lesion.

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FIG. 268 (A *Left*) Lymphosarcoma of the stomach. The deformity is obscured at this time. (B *Right*) Same patient. Mucosal relief study showing the deformity produced by the lesion.

tated hospitalization and transfusion. Operation revealed an ulcer of the greater curvature. The area was markedly indurated. A partial gastrectomy was done. Gross examination of the specimen showed that the crater was rather shallow and situated in the center of a hyperplastic mass (Fig 270 A). The microscopic diagnosis was lymphosarcoma.



FIG 271 (A, *Top*) Sarcoma of the stomach, simulating the appearance of polypoid adenocarcinoma (B, *Bottom*) Appearance of resected specimen

Roentgen examination (Fig 270 B) showed a well-rounded niche, extending from the greater curvature, pars media. Primarily because of its location, the lesion was considered to be malignant, and prompt resection was advised. It is interesting that one pathologist who first examined the slides, although recognizing the marked infiltration of the entire thickness of the gastric wall, chiefly by lymphoid cells, thought that the ulcer was not malignant. On review of the slides at Bellevue Hospital, the final diagnosis was lymphosarcoma.

A sarcoma of the stomach may also appear as a large bulky polypoid lesion, producing primarily an intraluminal area of translucency.

The patient, R. C., male, aged 49, gave a 4-month history of abdominal discomfort initiated by violent retching which lasted for a couple of days. Following this he noted pain in the lower half of the abdomen, severe in character, without definite relation to food intake and relieved by vomiting.

Physical examination of the abdomen revealed a firm movable slightly tender mass approximately 6 inches in diameter in the umbilical region.



Roentgenographic examination (Fig. 271 A) revealed a huge defect involving most of the distal half of the stomach mainly near the lesser curvature and characterized by an irregularly outlined translucent area. There was also a filling defect of the greater curvature pars pylori. The stomach was considerably enlarged, and at 6 hours there was a retention of three quarters of the barium meal. The evidence indicated a malignant tumor of the stomach with a polypoid mass producing the large area of intraluminal translucency.

Operation revealed a friable hemorrhagic mass involving about one quarter of the greater curvature, the anterior surface of the stomach and extending downward involving the entire area of the greater omentum. The mass was adherent to the anterior abdominal wall on the left side about 2 in. below the umbilicus. There were no enlarged lymph nodes found. A subtotal gastrectomy was done.

Microscopic examination. Specimen consists of a mass of tissue 30 x 15 x 6 cm. The bulk of the specimen is a solid pinlike shaped tumor, measuring 15 cm. across and 6 cm. thick. On section the tissue is white, friable, oozing a mucoid fluid. There are no definite cystic areas present.

Microscopic examination. For the most part the tumor is composed of moderate sized deeply staining round cells with poorly defined cytoplasm. At the base of the mucosa the tumor cells can be seen invading it. One of the two lymph nodes found shows metastatic invasion by this highly cellular tumor. There is no tendency to form tubules or alveoli.

Diagnosis. Undifferentiated sarcoma of the stomach with metastasis to one lymph node. Figure 271 B shows the gross appearance of the tumor.

Therefore in this case the sarcoma of the stomach had a roentgen appearance similar to that of a polypoid adenocarcinoma.

In the following case the deformity of the stomach was caused by a leiomyosarcoma.

C. J. male, aged 62. Four days before his admission to the hospital the patient had developed what he described as acute indigestion. He had crampy abdominal pain and diarrhea. He felt nauseated and forced himself to vomit. The stools were black and continued to be so. He had a chill with the onset



Fig. 272 Leiomyosarcoma of the stomach.

He had been losing weight for 3 months. There was no preceding history of digestive disturbance.

Physical examination revealed a bulging mass in the epigastrium which felt nodular and not very tender.

The autopsy report follows. On the anterior surface of the stomach midway between the lesser and the greater curvatures there is a large hole about 8 cm. in diameter, which is continuous with a large mass of firm whitish nodular tissue and adhesions. This mass is limited anteriorly by the anterior abdominal wall and the diaphragm and laterally by the anterior surface of the left lobe of the liver. Abdominal adhesions have surrounded this mass forming a sac in which the tumor is localized. On section, the margin of the perforation is firm white and gritty and almost the entire anterior wall of the stomach is thickened and the normal rugae distorted. The contiguous enlarged perigastric nodes are of the same firm white substance. There are about 30 cc. of thick, foul smelling brown fluid in the wall of the sac as well as a few flecks of barium. The pylorus admits one finger and is not obstructed.

Final diagnosis (including microscopic find

ings) was as follows leiomyosarcoma of the stomach with ulceration and perforation of the anterior wall, with metastases to the peritoneum, diaphragm, liver, perigastric and retroperitoneal lymph nodes and pericardium

Roentgen examination (Fig 272) revealed marked deformity of the proximal portion of the stomach with irregularly outlined translucent areas The barium appeared to extend beyond the confines of the greater curvature of the stomach into a walled-off area

It was believed that these findings might be explained on the basis of a malignant lesion of the stomach which had broken down with perforation into a walled-off cavity extragastric in location or that the mass might

have been originally extragastric with secondary ulceration and invasion of the stomach As noted at autopsy, the cause was a leiomyosarcoma of the stomach which had broken through the stomach and communicated with a large walled-off sac Although a diagnosis of a perforating malignant lesion of the stomach would be fully justified by the roentgenologic findings, there is nothing in the nature of the deformity to indicate that the lesion was a leiomyosarcoma

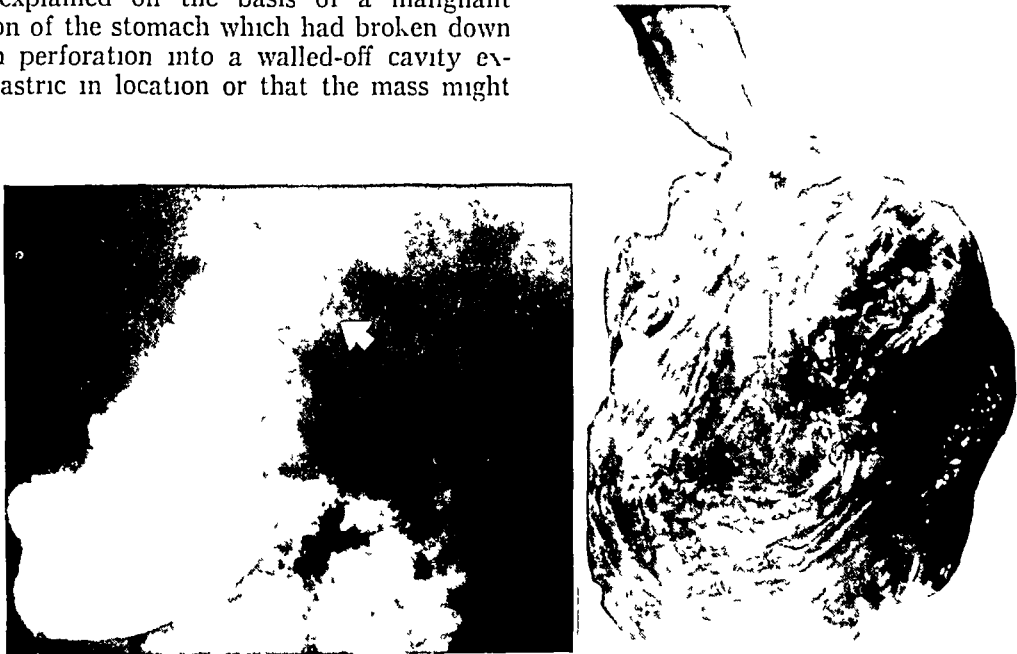


FIG 273 Leiomyosarcoma of the stomach (A, *Left*) Note the displacement of the stomach to the right, the irregularity of the contour of the greater curvature, and the large niche (B, *Right*) Autopsy specimen showing the gastric side of the tumor with 4 ulcers



FIG 273 (C, *Left*) Leiomyosarcoma of the stomach simulating the appearance of a benign tumor When the stomach is completely distended with barium the deformity produced by the tumor cannot be visualized (D, *Center*) As the result of compression the well-defined intraluminal translucent area produced by the tumor may be clearly seen (E, *Right*) Gross appearance of the excised tumor.

A leiomyosarcoma with ulcerations is shown in the case of A W, male aged 50

The patient gave a 6 month history of occasional vomiting after meals. This became progressively worse until several months later when he vomited after every meal. He then noted that his abdomen became swollen and he entered another hospital where a diagnosis of 'cirrhosis' was made. He lost 15 pounds during that period.

The essential findings on physical examination were

1 A protuberant abdomen with evidence of fluid

2 A large mass in the left upper quadrant extending to 4 fingers' breadth below the left costal margin. The liver was not palpable.

While under observation the ascites increased. Paracentesis yielded 8,500 cc of yellowish green fluid. This was repeated 2 weeks later when 8,000 cc of fluid was removed. Cell blocks of the ascitic fluid were negative. A third paracentesis 8 days later yielded 6,700 cc of fluid. Just prior to this procedure the patient complained of pain in the left upper quadrant which became worse. He suddenly vomited black watery material (positive for blood), went into shock and died within a few hours.

Roentgenographic examination (Fig 273 A) revealed evidence of a large mass in the left upper quadrant. The stomach was displaced to the right. There was a large niche at the proximal portion of the greater curvature with considerable irregularity of contour distal to it. The deformity of the greater curvature was indicative of an infiltrating lesion. Confirmation of these findings as well as a mucosal pattern study was planned but before this could be done the patient had the massive hemorrhage which was followed by death in a few hours. The real nature of the lesion was disclosed at autopsy.

On opening the abdominal cavity an unmeasured amount of gas is released. All the intestines are moderately adherent to one another by an easily separated green gray fibrinopurulent exudate. There is a large mass in the left upper quadrant which extends 6 cm below the left costal margin with adherent omentum on its surface. In addition there is adherent to the mass the liver, the spleen, the stomach and the hepatic flexure. There are 2,000 cc of bloody fluid in the peritoneal cavity.

G I Tract. The esophagus is normal. In the stomach on the greater curvature in the

cardia are four tremendous ulcerations, the largest measuring 4 cm in diameter. The largest ulcer opens directly by a fistulous tract into the center of the necrotic tissue which in turn apparently opens into the peritoneal cavity. This is the only source found to explain the peritonitis or the pneumoperitoneum. This is apparently the source of bleeding also although no definite vessel is found and there is bloody fluid in the stomach. About two thirds of the lesser curvature of the stomach is adherent to this tumor mass which on section is pink and necrotic except for areas near the stomach and near the direct extension to the liver which are rather firm and yellowish white. The entire mass, plus the stomach, weighs 1,800 Gm. The small bowel, the appendix and the large bowel are normal. The splenic flexure has been adherent to the inferior portion of the mass but is easily separated from it. There is no gross blood in the small or the large bowel.

Microscopic Examination. Stomach Ulcer and Tumor. The base of the ulcer is composed of tumor tissue which infiltrates beneath the submucosa. It is composed of interlacing bands of spindle shaped cells with oval, fusiform nuclei. There are numerous small thin walled vessels throughout. There is no rosette formation or clean cut palisading of nuclei. Some areas show numerous mitotic figures, and the growth is less orderly. Other sections of tumor show a similar growth with large areas of necrosis.

Trichrome and phosphotungstic acid stains reveal that the tumor is derived from smooth muscle fibers.

Lymph Nodes. One portal lymph node shows secondary tumor.

Diagnosis. Leiomyosarcoma of the stomach with direct extension to the liver, metastasis to lymph nodes, ulceration, perforation and hemorrhage.

Figure 273 B shows the gastric side of the tumor with the four areas of ulceration.

In reviewing the roentgenographic evidence the mass in the left upper quadrant with displacement of the stomach to the right was due to the leiomyosarcoma of the stomach. The deformity of the greater curvature was the result of infiltration by the tumor with ulceration within it. The niche high up on the greater curvature represented one of the ulcers on the gastric side of the tumor.

A leiomyosarcoma may simulate a benign tumor in its roentgenographic manifestations.



FIG 274 Neurofibroma of the stomach. The roentgenographic appearance is indistinguishable from an infiltrative carcinoma.

M. K., female, aged 51. The patient was admitted to the hospital with a history of tarry stools on numerous occasions. She had no abdominal pain. There was no weight loss. Physical examination of the abdomen was essentially negative. Operation revealed a tumor of the stomach about 5 cm in diameter and of smooth outline arising from the posterior wall of the distal portion of the stomach. A local enucleation of the tumor was done. It was the definite opinion of the surgeon that the tumor was benign, both before and after excision.

Pathologic examination: "Specimen consists of a globular-shaped tumor mass measuring 6 cm in diameter. The mass is rather soft and encapsulated. It is partly covered by an intact gastric mucosa. On section the tumor is grayish white in color and has a lobulated appearance. There is some interstitial hemorrhage present." From the gross appearance it was believed that the tumor was probably benign. However, microscopic examination revealed the tumor to be a leiomyosarcoma of the stomach. The slides in this case were studied by several pathologists in different institutions. The diagnosis of leiomyosarcoma was confirmed unanimously.

From the roentgenologic point of view this case is of considerable interest. When the stomach was completely distended with barium

there was no evidence of an intraluminal tumor (Fig 273 C). By means of compression a large, rounded, fairly sharply defined, translucent, intraluminal area was noted near the lesser curvature of the stomach at about the junction of the pars pylorus and the pars media (Fig 273 D). Based on this appearance a diagnosis was made of a benign tumor of the stomach. Figure 273 E shows the gross appearance of the excised tumor. When I was informed that the tumor had a sessile base I stated that a partial gastrectomy would have been preferable to the local removal because of the possibility of malignant changes which could be recognized only on microscopic examination, even though the tumor had the roentgen and gross characteristics of a benign lesion. As noted in the report of the microscopic findings the tumor actually proved to be malignant. At a subsequent operation a subtotal gastrectomy was performed.

Therefore, this case is of considerable interest for three main reasons: (1) It illustrates how easily even a large intraluminal tumor may be missed when the stomach is completely filled with barium; also, it shows the importance of mucosal pattern and compression studies. (2) Even when the roentgen criteria justify the diagnosis of a benign tumor, as they did in this case, microscopic examination may prove that the lesion is malignant. Therefore, no tumor, no matter how benign it may appear to be, can be viewed with complacency. (3) From the gross appearance the surgeon was convinced that the tumor was benign. Particularly because of the absence of a pedicle a more radical resection might well have been the procedure of choice at the time of the first exploration.

The following is a case of neurofibroma of the stomach.

Y. B., female, aged 65. The patient stated that in the preceding 9 years she had become progressively weak, accompanied by moderate weight loss. There was no pain or vomiting until a few months prior to admission to the hospital. She then developed epigastric pain and occasional blood-free vomiting.

Roentgenographic examination (Fig 274) revealed a diffuse narrowing of the stomach, rigidity of the contour and an absence of a normal mucosal relief pattern. The appearance was that of a diffuse, scirrhous malignant infiltration of the stomach. At operation the walls of the stomach were found to be hypertrophied. At the pylorus there was definite

thickening by a tumorous mass. The mass extended along the lesser curvature for a distance of about 3 in. and also along the greater curvature for about 2 in. The rest of the stomach wall appeared to be normal except for the marked hypertrophy. There were no palpable nodes in the gastrohepatic omentum.

A biopsy was taken. The pathologic report was neurofibroma of the stomach.

In reviewing the radiographic evidence, in the light of the pathologic findings, there appeared to be no features which distinguished the deformity of the stomach from an infiltrating carcinoma.

REFERENCES

1. Rickles, J. A. Multiple carcinomas of the stomach, *Surgerv* 19 229, 1946.
2. Marvin, C. P., and Walters, W. Leiomyosarcoma of the stomach, *Arch Surg* 57 62, 1948.
3. Neel, H. B. Multiple carcinomas of the stomach and choledocholithiasis in a patient with pernicious anemia. *Minnesota Med* 31 1077, 1948.
4. Kadrnka, S. and Sierro, A. Le sarcome primitif de l'estomac, diagnostic clinique et radiologique, *Arch mal app digest* 23 51, 1933.
5. Balaban, J. Zur Frage des primären Magensarkoms. *Fortschr Geb Röntgenstrahlen* 49 513, 1934.
6. Symmers, D. Certain unusual lesions of the lymphatic apparatus, including a description of primary Hodgkin's disease of the spleen and a case of gastro intestinal pseudoleukemia, *Arch Int Med* 4 218, 1909.
7. Wells, H. G. and Mayer, M. B. Pseudo-leukaemia gastro intestinalis, *Am J M Sc* 128 837 1904.
8. Pearson, F., Stasney, J. and Pizzolato, P. Gastro intestinal involvement in lymphatic leukemia, *Arch Path* 35 21 1943.
9. Svab, V. Ein Fall von aleukämischer Lymphadenose des Magens im Röntgenbild, *Med Klin* 26 1922, 1930.
10. Mead, C. H. Chronic lymphatic leukaemia involving the gastro intestinal tract. *Radiology* 21 351, 1933.
11. Koch, C. E. Leukämische und pseudo-leukämische Wandveränderungen des Magens im Reliefbild. *Fortschr Geb Röntgenstrahlen* 48 271 1933.
12. Kaznelson, P. Über einen Fall von Nischenbildung und Pylorusstenose in Folge Lymphogranulomatose des Magens. *Wien Arch inn Med* 7 117 1923.
13. Singer, H. A. Primary isolated lymphogranulomatosis of the stomach. *Arch Surg* 22 1001 1931.
14. Koenig, E. C., and Culver, Gordon J. Hodgkin's disease involving the stomach—report of two cases, *Am J Roentgenol* 46 827, 1941.
15. Jungmann, H. Hodgkin's disease of the stomach. *Brit J Radiol* 16 386, 1943.
16. Pirkey, E. L., and Roberts, S. M. Diagnosis of primary Hodgkin's disease of the stomach. *Radiology* 52 75, 1949.
17. Sandwick, H. Hodgkin's disease involving the stomach. *Gastroenterology* 15 135, 1950.
18. Wallhauser, A. Hodgkin's disease. *Arch Path* 16 522, 672, 1933.
19. Holmes, G. W., Dresser, R., and Camp, J. D. Lymphoblastoma: its gastric manifestations with special reference to the roentgen findings. *Radiology* 7 44 1926.
20. Ruggles, H. E., and Stone, R. S. Lymphoblastoma involving the stomach: roentgenographically considered, *California & West Med* 33 486 1930.
21. Ende, Norman, Daron, P. B., Richardson, L. K., Raider, L., and Ziskind, J. Plasma cell tumor of the stomach. *Radiology* 55 207, 1950.
22. Large, H. L. Jr., McChord, William, and Neel, J. B. Gastric tridermal teratoma in infancy, *JAMA* 149 824, 1952.
23. Steinhilber, F. Ueber eine seltene Form von Amyloid und Hyalin Infiltration am Circulations- und Digestionsapparat, *Ztschr Klin Med* 45 375, 1907.
24. Clausen, A. Fall von röntgenologisch wahrnehmbarer Magenamyloidose. *Fortschr Geb Röntgenstrahlen* 51 528 1935.
25. Koletsky, S. and Stecher, R. M. Primary systemic amyloidosis. *Arch Path* 27 267 1939.
26. Gotttron, H. Systematisierte Haut Muskel Amyloidose unter dem Bilde eines Skleroderma amyloidosum. *Arch Dermat Syph* 166 584 1932.
27. Golden, A. Primary systemic amyloidosis of the alimentary tract, *Arch Int Med* 75 413, 1945.

- 28 Shipps, F C. and Brannan D D Roentgenological findings in amyloidosis of stomach A case report *Am J Roentgenol* 68 204, 1952
- 29 Von Recklinghausen, F Ueber die Multiplen Fibrome der Haut und ihre Beziehung zu den multiplen Neuomen, pp 14 and 15 Berlin, A Hirschwald 1882
- 30 Banks, B M Neurofibroma of the stomach, *Gastroenterology* 14 158, 1950
- 31 Baty, J A Gastric neurilemmoma *Brit J Surg* 39 251, 1951
- 32 Smith, W H Neurofibroma of the stomach, *Brit J Radiol* 25 110, 1952
- 33 Bock, Karl Ein Beitrag zum Neurofibromatose des Magens, *Fortschr Geb Rontgenstrahlen* 73 101, 1950
- 34 Canney R L Neurogenic tumors of the stomach, *Brit J Surg* 36 139 1948
- 35 Gillespie, H W Neuroepithelioma of the stomach, *Brit J Radiol* 20 433, 1947
- 36 Truglio, Vincenzo A contributo alla conoscenza delle complicazioni gravi della neurofibromatose gastro-intestinale, *Sicilia med* 6 57, 1949
- 36A McKusick V A Boeck's sarcoid of the stomach with comments on the etiology of regional enteritis *Gastroenterology* 23 103, 1953
- 37 St John, F B Swenson, P C, and Harvey H C An experiment in the early diagnosis of carcinoma, *Ann Surg* 119 225, 1944
- 38 Rambach H Über die Entwicklung von Magenkrebs bei der perniziösen Anämie, *Monatsschr Krebsbekampf* 4 201, 1936
- 39 Zancan, B Sui rapporti tra anemia perniziosa e carcinoma gastrico, *Minerva med* 2 645, 1937
- 40 Doehring, P C. and Eusterman, G B Association of pernicious anemia and carcinoma of the stomach, *Arch Surg* 45 554, 1942
- 41 Cotti, L Elievi casistico bibliografici e contributo personale in merito alla conoscenza dei rapporti fra anemia perniziosa e carcinoma gastrico, *Haematologica* 19 939, 1938
- 42 Rigler, L G. Kaplan H S. and Fink, D L Pernicious anemia and the early diagnosis of tumors of the stomach, *J A M A* 128 426, 1945
- 43 Weinberg F Achylia gastrica und perniziose Anämie *Deutsche Arch Klin Med* 126 447, 1918
- 44 Meulengracht, E The heredity factor in pernicious anemia, *Am J M Sc* 169 177, 1925
- 45 Gram H C Further observations on a family showing many cases of pernicious anemia, *Acta med scandinav* 34 107, 1930
- 46 Conner, H M Hereditary aspect of achlorhydria in pernicious anemia, *J A M A* 94 606, 1930
- 47 Thiele, W Perniziose Anämie und Magencarcinom unter besonderer Berücksichtigung ihres familiären Auftretens, *Klin Wchnschr* 15 921 1936
- 48 Bloomfield, A L. and Pollard, W S The fate of people with unexplained gastric anacidity *J Clin Investigation* 14 321 1935

Syphilis and Tuberculosis of the Stomach

SYPHILIS OF THE STOMACH

The diagnosis of syphilis of the stomach is fraught with difficulty. Both the frequency of the disease and the pathologic criteria for diagnosis have been a matter of considerable dispute. One need hardly be surprised then, to learn that the roentgen method which is based primarily on defects of contour produced by the ingestion of a contrast medium, also provides no sharply defined diagnostic criteria.

Theoretically the actual finding of *Spirochaeta pallida* in the diseased tissue of the stomach should be considered as essential for absolute proof of the presence of a syphilitic etiology. If that were the case many of the organic derangements of the stomach which have been reported as syphilitic would have to be eliminated from consideration.

What has made the positive identification of syphilis of the stomach a matter of controversy are the following facts. First a positive Wassermann reaction in the presence of a roentgenologically demonstrable gastric deformity can, of course not be considered as evidence of a causal relationship. Obviously, an independent gastric lesion and syphilis may coexist. Secondly therapeutic response to antisiphilitic therapy, with diminution of the degree of gastric deformity or its complete disappearance likewise offers at best only suggestive proof. The gastric lesion may have been only apparent and due to spasm of intrinsic origin. The stomach is notoriously a fertile field for the play of spasm. The deformity may be persistent in character simulating

organic infiltration, and yet be due to an ulcer which, during the course of anti-syphilitic treatment, undergoes healing with secondary disappearance of the concomitant spasm although the ulcer bears no relation to the associated syphilis. The improvement in gastric contour may then be laid at the door of the treatment involved. Moreover by riding the underlying although independent syphilitic infection the general well being of the patient is aided and the healing of his gastric disorder thereby quickened. Chronic gastritis and pyloric hypertrophy of benign origin may produce deformities of contour which, in the presence of a positive Wassermann, may also suggest syphilis.

Finally, in the study of the resected specimen the pathologic criteria are often of such a doubtful nature as to lead to disagreement as to what constitutes acceptable evidence unless the *Spirochaeta pallida* is actually demonstrated in the stained specimen. A somewhat similar problem confronted pathologists in the diagnosis of the true nature of linitis plastica. Brinton¹ to whom we are indebted for the introduction of the term, believed the condition to be benign. Only painstaking search throughout the entire extent of the infiltration disclosed the true nature of the disorder as belonging to the class of malignant disease.

Possibly some of the lesions considered syphilitic because of the failure to find cancer cells may fall into this classification. Encouragement in this belief may result also from the slow growing character of the infiltration and its compatibility with a fair degree of health for a number of years.

These factors probably explain the marked discrepancy in the figures regarding the frequency of this lesion emanating from various large clinics. On the other hand, there is no doubt that, although the condition is rarer than many believe, authenticated cases are on record.

That syphilis of the stomach is rare is amply demonstrated by a survey of the contributions to the pathology of the disease.

Thus Jonathan Hutchinson in his textbook in 1887 stated that he knew of no museum specimens or of any published observations of syphilis of the stomach. So rare did Cornil² consider syphilis of the stomach to be that he recommended that any case suspected of being such should be subjected to the most rigorous critique. However, he did report in great detail his finding of a gumma of the stomach during the course of an autopsy in the case of a woman who also had a gumma of the liver. The rarity of syphilis of the stomach was also recognized by Aschoff³ and, like Cornil, he stressed the importance of examining any suspected lesion very critically before accepting it as a sequela of specific disease.

The difficulty in differential diagnosis was accentuated by Hartwell⁴ who described one of our cases at Bellevue Hospital. Roentgen examination disclosed a tubular narrowing of the distal half of the stomach which preoperatively had been reported as carcinoma. Gastric analysis showed no free hydrochloric acid. The Wassermann was 4 plus. The lesion was resected, and examination showed an irregular, serpiginous, superficial ulceration of the posterior wall. The histologic characteristics were those of ulcer without any suggestion of syphilis, although the findings were similar to those in cases reported in the literature as syphilis of the stomach.

The difficulty of determining whether a gastric lesion is syphilitic on the basis of the histologic findings is emphasized by Williams and Kimmelstiel⁵ in their report of

a case as well as a survey of 8 other cases which had previously been reported from their clinic. They emphasized the fact that none of the microscopic criteria are in themselves pathognomonic but that these findings assume significance only in connection with clinical and serologic evidence of luetic infection.

Our own experience at Bellevue Hospital indicates that syphilis of the stomach must be an extremely rare disease. Symmers reported a case in the autopsy material at Bellevue Hospital that he considered as syphilitic in nature. This patient, a white male, aged 32, showed at autopsy a syphilitic liver with multiple gummata, not only in the liver, but also in the mesenteric, pancreatic and perigastric lymph nodes. There was a syphilitic aortitis. The stomach showed extensive ulcerative lesions. Histologic study showed evidence characteristic of syphilis, such as chronic productive inflammatory changes attended by miliary gummas, endarteritis obliterans, and plasma and round-cell infiltration.

On rare occasions since this original report there have been gastric lesions apparently of syphilitic origin. The details of several of these cases including the roentgen findings are to be found at the conclusion of this chapter.

In contradistinction to the experience of most observers, Eusterman⁶ described 93 cases as having come under observation at the Mayo Clinic. In the advanced stage, the appearance of the stomach was similar to that seen in linitis plastica. Some showed hourglass contraction. A number of examples of improvement in the roentgen appearance of the gastric deformity, considered to be syphilitic in origin, occurred after treatment by antisyphilitic measures.

Because of the difference in opinion as to the relative frequency of syphilis of the stomach and the criteria justifying this diagnosis, the finding of *Spirochaeta pallida* would be of enormous value in demonstrating the syphilitic character of such a lesion.

Such findings were reported by McNee⁷

in the case of a man 57 years old with a history of abdominal pain, anemia and considerable loss of weight. A hard irregular mass, the size of a hen's egg, was palpable in the epigastrium. There was an absence of free hydrochloric acid in the gastric contents. A diagnosis of carcinoma was made. No Wassermann was done because it was believed that the patient had a malignant growth. At autopsy nothing was found to suggest syphilis. The stomach was greatly thickened. The lesser curvature of the stomach showed considerable ulceration from just below the cardia to the pylorus. The ulcer was of irregular shape with greatly thickened borders. Histologic examination showed round cell infiltration along the blood vessels. No giant cells were present. Examination by the Levaditi silver method showed great numbers of spirochetes penetrating into the actively growing granularomatous tissue. Pictures of the gross and microscopic specimens, as well as the one showing spirochetes, accompany the article. This was the first example of spirochetes in a gastric lesion which had been recorded in the entire literature of gastric syphilis.

It seemed quite strange that this case should have been the first in which the *Spirochaeta pallida* was demonstrable in a gastric lesion and a clue to the apparent mystery is furnished by the work of Simmonds⁸ who in 1908 described the occurrence of spirochetes in broken down cancers of the mouth, the esophagus and the stomach. Occasionally he found them also in broken down cancers of the skin, the uterus and the intestine, but never in the prostate, the urinary bladder, the gallbladder or the kidneys. Thus the spirochetes occurred only in those locations which might come in contact with saliva; the spirochetes were invariably absent in those locations which could not be reached by saliva. Simmonds therefore believed that these spirochetes originated from organisms present in the mouth. They are of no clinical importance except in so far as these organisms might be mistaken for those of syphilis.

This work was verified by Luger and Neuberger,⁹ who also showed that spirochetes may be found in broken down carcinomas of the stomach. The occurrence of spirochetes and fusiform bacilli in the mouth has long been established. In a large number of pathologic processes these may increase enormously. Luger and Neuberger examined 10 cases of carcinoma of which 9 were carcinomas of the stomach and 1 a carcinoma of the esophagus. In 8 of the gastric carcinomas, as well as in the case of carcinoma of the esophagus examined by the streak method immediately after operation or autopsy, they revealed the presence of many spirochetes. The more advanced the breaking down process of the tumor, the greater was the number of spirochetes found. There was no evidence of the presence of spirochetes in the normal stomach of man. Luger and Neuberger also examined the fasting contents in 180 patients. Darkfield examinations revealed the presence of spirochetes in 22 out of 28 gastric carcinomas. In 3 out of 45 cases of ulcer, a very small number of spirochetes were found. They found 5 types of spirochetes but none that could be considered as characteristic for carcinoma.

The spirochetes shown in McNee's case were evidently of the type reported by Simmonds and Luger and Neuberger and were not *Spirochaeta pallida*. Singer and Dyas¹⁰ also succeeded in demonstrating spirochetes in ulcerated areas which were similar if not identical with those described by Vincent as occurring in tissue which is the site of saprophytic infection.

The spirochetes reported by Harris and Morgan¹¹ as having been found in a resected tumor of the stomach appear to meet criteria which would justify their being considered as being due to syphilis. A suspension of involved tissue was injected into the right testicle of two rabbits and, on the forty eighth day, a syphiloma appeared. In one case there was an associated chancre of the scrotum. Darkfield examinations of the lesions as well as tissue stained by the

Levaditi method showed typical *Spirochaeta pallida*

Another aid in the diagnosis of the syphilitic nature of a gastric lesion is the determination of the therapeutic response to antisyphilitic treatment and its intractability to all other measures, as emphasized by Fenwick.¹² It is this feature which has been a guide in the diagnosis of a syphilitic affection of the stomach on the basis of roentgen evidence of diminution in the degree of deformity or its complete eradication.

Roentgen Diagnosis of Syphilis of the Stomach. In a condition in which competent pathologists are not in agreement as to the exact nature of the anatomic features which would justify a diagnosis of gastric syphilis, it is obvious that roentgen criteria must be of an even less convincing character. There are no changes in the roentgen appearance of the stomach which can be considered as peculiar to syphilitic infection and clearly differentiable from the various types of deformity produced by malignant infiltration. However, tubular narrowing of the pars pylorica, was present in those cases I examined which histologically showed strong evidence of a syphilitic etiology.

In the well-documented case reported by Palmer and his associates¹³ it is noteworthy that neither the roentgen nor the gastrosopic examination offered any clue to the luetic nature of the gastric lesion. The resected specimen disclosed a shallow serpiginous prepyloric ulcer, with histologic evidence compatible with syphilitic etiology.

The main reliance from the purely roentgenologic point of view has been the evidence of a successful therapeutic response, with diminution in the degree or complete disappearance of the original deformity. On this basis a fairly large number of reports have appeared in the literature. In 1911 B  cl  re and Bensaude¹⁴ examined a 54-year-old patient with a history of syphilitic infection and a gumma of the left leg who had complained of gastric symptoms for

11 years. Roentgen examination revealed an hourglass stomach. Under specific treatment the hourglass deformity gradually disappeared until the final roentgenogram showed an essentially normal stomach. Additional reports of gastric deformities altered by antisyphilitic measures were those of Meyers,¹⁵ Bockus and Bank¹⁶ and Moore and Aurelius.¹⁷

Particularly important in a comparative study of the effectiveness of antisyphilitic measures in the reduction of the degree of deformity in the roentgenogram is the employment of a rigid technic, the following precautions are considered advisable. First, a comparative study in the degree of deformity should be made after a lapse of several days or weeks before the institution of antisyphilitic treatment. Considerable change in the degree of deformity may then be noted, due to relaxation of spasm, which might otherwise be attributed to the effect of the antisyphilitic treatment. Moreover, abnormalities of contour secondary to an underlying gastritis or ulcer may show considerable diminution after medical management for these conditions even though a positive Wassermann is present.

Secondly, in comparing the effect of antisyphilitic measures on the appearance of a deformed stomach, it is essential that the roentgen technic be identical as regards the amount of barium administered and the position of the patient. Even in a series of films taken during the course of a single examination the gastric deformity may show considerable variation in the different films because of variations in the degree of filling, the exact position of the patient and the transient factor of momentary spasm.

For improvement in a gastric deformity to be considered as having resulted from antisyphilitic treatment, the change must be one that cannot be accounted for by any of the above-named factors and occurring only after such treatment.

Finally, one may summarize the situation as follows. Syphilis of the stomach is probably a rare disease. For the most part, the

roentgen manifestations may be indistinguishable from infiltrative processes of a nonspecific or malignant nature. Clinically, the condition may be suspected in the presence of other evidence of syphilitic infection and a successful therapeutic response, with disappearance of the gastric deformity, provided that spasm and other organic lesions such as ulcer and chronic gastritis can be eliminated and a proper technic for comparative study has been followed.

Cases of probable syphilis of the stomach are described at the end of the discussion on tuberculosis of the stomach.

TUBERCULOSIS OF THE STOMACH

Tuberculosis of the stomach is also a very rare disease, although comparatively frequent elsewhere in the intestinal tract. Here the diagnostic criteria are a matter of considerable difference of opinion in the absence of the demonstration of the tubercle bacillus in the gastric tissue.

Rokitansky¹⁸ emphasized the fact that tubercular involvement of the stomach is very rare, and the primary form is almost unknown.

Although reports of tuberculosis of the stomach appeared in the literature for many years prior to the discovery of the tubercle bacillus by Koch in 1882, the first case of this nature in which the newly discovered organism was actually demonstrated in the wall of a gastric ulcer was that by Coats¹⁹ in 1886 who recognized the fact that while the intestine is frequently the seat of tubercular ulceration "the stomach is so with excessive rarity." He reported one case, of a patient with advanced pulmonary tuberculosis whose stomach showed a considerable number of ulcers of a tuberculous nature. Tubercles were present on the peritoneal surface of the stomach becoming more marked near the lesser curvature. Abundant tubercle bacilli were found both in the ulcers in the stomach and in the tubercles and glands outside the stomach. The actual specimen showing the bacilli

was placed under the microscope and exhibited before the Medico-Chirurgical Society of Glasgow, March 5, 1886. Coats believed the reason for the extreme rarity of tuberculosis of the stomach to be the fact that there are no closed follicles in the stomach as there are in the intestine.

Musser⁹ examined the stomach of a Negro 44 years of age who had died of pulmonary tuberculosis. There was an ulcer of the lesser curvature of the stomach extending down to the peritoneum. At the floor of the ulcer small tubercles could be seen. Nodules in the region of the ulceration showed caseous material on section. The ulcer had the appearance of having resulted from the breakdown of a large, submucous, cheesy mass. The histologic appearance was characteristic of tuberculosis and the tubercle bacillus was demonstrated.

In Dewey's¹ case the patient at autopsy showed ulcerative tuberculosis of both apices of the lungs, caseous tuberculosis of the tracheobronchial lymph nodes and ulcerative tuberculosis colitis as well as tuberculosis of the stomach. Grossly the stomach showed ridges and polypous elevations mainly about the lesser curvature. The diagnosis was based on microscopic examination of the lesions as well as the finding of tubercle bacilli in large numbers. Giant cells were found in nearly every section. Rows of lymphocytes extended along the muscularis mucosae and in the submucosa.

According to Broders, who made an exhaustive analysis of the literature, tuberculosis of the stomach may manifest itself as single or multiple ulcers, solitary tubercles, solitary tubercle, pyloric stenosis (resulting either from an old tuberculous ulcer or from the pressure of enlarged lymph nodes situated about the pylorus), single or multiple tumors or nodules, or as a lymphangitis. Broders described in detail the only case he encountered out of a series of 2501 gastric operations at the Mayo Clinic from 1912 to 1915. Including the case in his own personal experience, he

recorded a total of 49 cases, of an apparently positive nature, of tuberculosis of the stomach. As a criterion of a positive case he demanded the demonstration of a histologic picture of tuberculosis and the presence of the tubercle bacillus in the lesion. In only 4 cases was the gastric tuberculous lesion considered to be primary. In Brodeur's case there was recent tuberculosis of both lungs. The stomach exhibited three ulcers of the pylorus. Microscopic study revealed typical tubercles and a few tubercle bacilli in the depths of the ulcers.

Roentgen Findings in Tuberculosis of the Stomach. The difficulty in differentiating tuberculosis of the stomach from malignant infiltration is well illustrated by the detailed report of Pohl,²³ who described the case of a patient 61 years of age who had gastric anacidity and evidence of tubercular involvement of the lungs. Roentgen examination showed an irregularity in the outline of the stomach, with localized areas of destruction of the mucosal folds. The roentgen diagnosis was carcinoma of the stomach, although the possibility of tuberculosis was considered because of the fact that there appeared to be no great change in the roentgen picture at the end of 2 years and because of the lesion in the lungs. Autopsy revealed extensive ulceration and infiltration limited mainly to the submucosa. The outer surface of the stomach, as well as the parietal peritoneum, were studded with milium tubercles. Tubercular ulcers in the ileum and the descending colon were also present. Histologic examination showed a tuberculous infiltration of the wall of the stomach with tubercles and acid-fast bacilli. Animal inoculations were positive for tuberculosis. Examination of the lungs showed a chronic cavernous tuberculosis. An examination of the roentgen evidence leads one to agree with the author that differentiation from carcinoma was impossible. (Neither the gross nor the microscopic appearance of the specimen accompanies the article.)

Other cases reported as tuberculosis of

the stomach that were examined roentgenologically were those of Pop and Hanganutan²⁴ and Renander.²⁵ In none of these cases was a preoperative diagnosis made, the appearance in the roentgenogram being that of malignant infiltration.

Except for the report by Pohl, the others lack complete confirmation in that there is no record of the demonstration of tubercle bacilli.

The nonspecific character of the roentgen deformity in gastric tuberculosis has been confirmed by a number of reports (Morris,²⁶ Ostrum and Seibel,²⁷ and Sherman and Johnston²⁸).

From the roentgenologic standpoint, therefore, there are no established criteria upon which a diagnosis of tuberculosis of the stomach may be made with any reasonable degree of assurance. However, as will be described in connection with the case of tuberculosis of the stomach reported at the end of the chapter, it is possible that a deformity of the pylorus associated with sinus tract formations emanating from the involved segment may be a significant diagnostic manifestation of the disease.

Illustrative Cases. The following is a case which was diagnosed as either syphilis or tuberculosis of the stomach on pathologic examination.

C. C., colored female, aged 24. This patient gave a history of pain after meals and vomiting during the preceding 2 years. She had lost 27 pounds. Vomiting occurred 2 hours after meals and relieved her pain. She felt better on a milk diet and experienced neither pain nor vomiting during the month preceding her admission to the hospital. Physical examination revealed a mass and tenderness in the epigastrium. The blood and spinal Wassermann were both negative.

Operation showed an extensive ulcer of the pyloric antrum extending from the incisura to the sphincter. The regional lymph nodes were enlarged but not particularly hard. Partial gastrectomy was performed. Three months later she was symptom-free and had gained 33 pounds.

The following was the pathologic report: "Specimen consists of a portion of a stomach with a small amount of omentum and two

lymph nodes which do not present any evidence of metastases. Serosa is not injected, pyloric opening admits tip of little finger. On section wall is thickened, submucosa edematous, mucosa thickened. There is a spherical fresh ulceration, $1\frac{1}{2}$ inches wide and extending for $1\frac{1}{2}$ inches from the pyloric opening encircling from the pyloric margin into the stomach cavity for a distance of 1 inch.

"Microscopic examination shows scattered throughout the wall of the stomach, particularly among the muscle fibers innumerable large and small, circumscribed or diffuse collections of round and plasma cells. In many instances these collections are arranged in the vicinity of blood vessels either circumferentially or eccentrically. In no instance however are the blood vessel walls noticeably thickened nor is there any noteworthy evidence of endothelial proliferation. Among the lymphoid and plasma cells, on occasions are to be made out solitary giant cells of the Langhans type without epithelioid reaction in the immediate vicinity. In still other instances the giant cells are associated with the overgrowth of epithelioid cells of the type commonly encountered in tubercles and gummata.

In one slide there is a very definite, circumscribed formation attended by central coagulation necrosis, arranged radially to which are ill formed epithelioid cells while at the periphery are vast numbers of round cells, many of them of the plasma cell type, others lymphocytic. The lesion represents undoubtedly, either a miliary tubercle or a miliary gumma. It is impossible histologically, with ordinary stains such as hematoxylin and eosin to differentiate between them. Sections are now being stained for spirochetes to exclude syphilis and for tubercle bacilli to exclude tuberculosis. It is suggested that a provocative Wassermann be done.

"In arriving at a histologic diagnosis in the present instance one meets with difficulties which are practically, for the time being, at least, insurmountable for the reasons already described. As far as our experience in this laboratory is concerned, syphilitic ulcers of the stomach are extremely rare, only one in dubitable case having been encountered among almost 17 000 autopsies. This experience however is not in keeping with that of certain published statements where syphilitic lesions of the stomach are rather nonchalantly



FIG. 275 A. Annular pyloric deformity, probably due to syphilis.

referred to as 'unusually common' In this laboratory we have once or twice suspected tuberculosis of the stomach but have never been able actually to demonstrate its tuberculous nature and as far as I know, genuine tuberculous ulcers of the stomach have never been absolutely proved to exist'

These suggestions of Doctor Symmers were carried out Neither spirochetes nor tubercle bacilli were found by special stains Wassermanns and provocatives were all negative The diagnosis remained either tuberculosis or

syphilis of the stomach, with the probability in favor of syphilis of the stomach

Roentgen examination (Fig 275 A) revealed a defect in the prepyloric region, which was most marked at the greater curvature The preoperative roentgen diagnosis was "malignant infiltration" As determined on pathologic examination, the lesion was either of syphilitic or of tuberculous origin, with the probability that the lesion was syphilitic The pathologic appearance of the specimen is shown in Figure 275 B, C, D and E



FIG 275 (B, *Top*) Same patient as is shown in Figure 275 A Excised stomach wall showing an annular ulcer and a polyp at the pyloric sphincter (C, *Bottom*) Same patient Low-power microphotograph (Summar lens 35 mm) of the edge of the ulcer Note the inflammatory base with thickened submucosa and infiltration beneath the mucosa adjacent to the ulcer



The following is an example of annular narrowing of the pyloric region, which was considered to be of probable syphilitic origin

M R, female, aged 31 The patient gave a 2 year history of epigastric pain and vomiting occurring about 1/2 to 1 hour after meals On several occasions small amounts of blood were present in the vomitus She had lost 25 pounds in the year preceding her admission to the hospital There was a history of syphilis, for which she had received treatment

At operation, a rather soft, circumscribed mass which almost completely constricted the pyloric ring was found There were no enlarged nodes and the mass did not have the appearance of either an ulcer or a carcinoma A partial gastrectomy was done

The report of the gross examination was as follows The serosal surface is smooth and glistening On the mucosal surface on the greater curvature just proximal to the pylorus there is an area of ulceration measuring 1.5 cm in diameter and from 0.5 to 0.75 cm in depth The stomach wall beneath the lesion is white and firm measuring 2 cm in thickness There is an enlargement of attached omental lymph nodes

Microscopically, the lesion was that of a cellular and moderately well vascularized chronic productive inflammatory process

characterized by the presence, in places, of focal lesions, which were well circumscribed by connective tissue and which consisted largely of lymphocytes which centrally were in the process of necrosis In addition, the connective tissue supported numbers of vascular channels the walls of which were thin and were surrounded by mantles of lymphocytes, the so called perivascular lymphocytic infiltration The areas composed of necrotic lymphocytes were highly suggestive of gumma

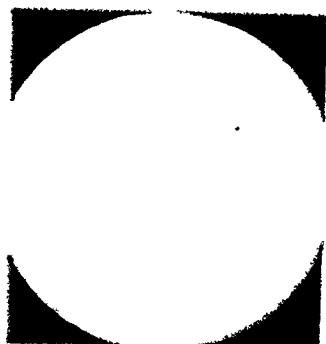
Figure 276 A shows the gross appearance of the resected specimen Figure 276 B shows a circumscribed area suggesting a gumma Figure 276 C (low power) shows perivascular lymphocytic infiltration Figure 276 D (high power) shows slight thickening of the vessel wall with rich infiltration of lymphocytes and plasma cells

Roentgen examination (Fig 276 E) showed the stomach to be of markedly increased size The pyloric antrum was narrowed, giving the appearance of a tubular structure Because of the tubular narrowing of the pyloric antrum the possibility was suggested that we might be dealing with a luetic lesion

The luetic background of this comparatively young Negroess (age 31), the peculiar tubular appearance of the pyloric antrum in the roentgenogram and the anatomic findings make the diagnosis of syphilis of the stomach strongly probable



FIG 275 (D Left) Same patient as is shown in Figure 275 A B and C Microphotograph low power showing round cell infiltration giant cell formation and fibrous connective tissue in the submucosa adjacent to the base of the ulcer Note the glandular tubules in the depth of the mucosa at the top (E Right) Microphotograph high power, to show perivascular infiltration by round cells and plasma cells



small amount of barium in the stomach at 6 hours

The evidence is that of annular pyloric narrowing of organic origin. The exact nature of the organic process producing this narrowing

cannot be determined on the basis of roentgen findings. Such an appearance can conceivably be due to a malignant process even though in this case careful pathologic study showed the lesion to be benign and the histologic findings



FIG. 276 E. Same patient as is shown in Figure 276 A D. Note the tubular narrowing of the pyloric antrum.

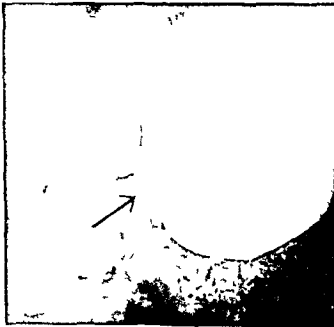


FIG. 277. Annular narrowing of the pylorus probably due to syphilis.

were those of either miliary gummata or tubercles

The next case is included only because the gross appearance of the lesion was suggestive of syphilis, and because of the difficulties involved in arriving at a diagnosis

J A, female, aged 40 The patient gave a comparatively brief history of from 7 to 8 days of nausea, vomiting of coffee-grounds material and localized epigastric pain of increasing severity Three days before her admission to the hospital, she vomited bright-red blood About 1 year previously she had had an appendectomy and a cholecystectomy Physical examination of the abdomen was essentially negative Gastric analysis showed an absence of free hydrochloric acid The Wassermann and the Kahn reactions were both positive She had a skin lesion which was diagnosed by the dermatologist as secondary syphilis

At operation "The distal third of the stomach was the site of a large serpiginous ulcer, which measured 3 cm across This ulcer had a fibrous-looking base, but the stomach surrounding it had a gelatinous appearance The

entire lesion was soft and elastic in places, was not stony hard and did not resemble a carcinoma in texture There was no evidence of carcinoma anywhere in the abdominal cavity Liver, spleen and kidneys were normal The stomach was slightly dilated The lesion was felt to have the possibility of being malignant, and for this reason a resection was performed "

The report of the macroscopic examination was as follows 'On opening the stomach, there is a large, serpiginous, rhomboid-shaped ulcer above the pyloric ring, measuring 5 cm x 2.5 cm with its long axis transverse The base of the ulcer is on the lesser curvature It has a glistening gray-white base, and its edges are sloping and well demarcated There is a small, elevated hemorrhagic area in the center of the ulcer The stomach wall in the area of the ulcer is thickened, but the various layers of the stomach are well outlined "

The report of the microscopic examination follows 'The floor of a broad but relatively shallow ulcer is seen It is covered by a ribbon of fibrinopurulent exudate Beneath this is a broad zone of vascular and cellular granulation tissue which extends into the submucosa It also contains strands of the muscularis mucosae, but the continuity of the latter is

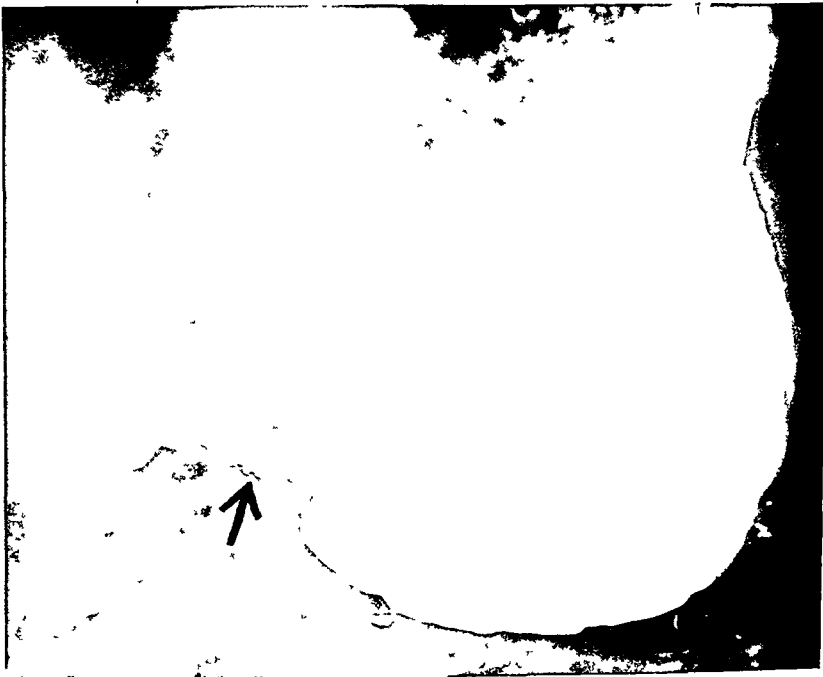


FIG 278 Serpiginous ulcer of the pylorus (syphilis?) Because of the annular narrowing of the pylorus and the irregular contour, the preoperative roentgen diagnosis was carcinoma

broken in many places. The newly formed vascular channels in the granulation tissue are directed toward the base of the ulcer. There is an intense cellular infiltration of lymphocytes, plasma cells and eosinophils, and this extends well beyond the ulcer base into the intact mucosa and submucosa at either edge. The edges of the ulcer are sloping, and the mucosal glands are irregular and appear to be regenerating to some extent. More deeply the submucosa is greatly thickened by edema fluid and connective tissue proliferation but is much less cellular. Here too this change extends well beyond the limits of the ulcer. In it and also in the thickened subserosal layer, numerous perivascular infiltrations of lymphocytes and plasma cells are found. The muscularis itself, although somewhat thinned out, is partly invaded by cellular granulation tissue but at no point is completely disrupted. At no place is there caseation or definite evidence of gumma formation. No multinucleated giant cells are seen.

The diagnosis was ulcer of the stomach. "Note. The gross appearance of the lesion is suggestive of syphilitic ulcer."

Roentgen examination (Fig. 278) revealed considerable irregularity of the lesser and greater curvatures of the stomach in its distal half. The prepyloric area appeared narrowed and there were a number of small rounded translucencies mainly near the deformed greater curvature. The appearance was interpreted as being due to a carcinoma. As noted however in the pathologic report a large serpiginous ulcer was found occupying the pylorus without any evidence of malignancy. Although the gross appearance of the lesion suggested to the pathologist that it might be a syphilitic ulcer, the microscopic findings did not clearly support this opinion. It is interesting however to note (1) that the roentgen deformity and the appearance of the ulcerating lesion were both unusual, (2) that there was an absence of free hydrochloric acid in the fractional analysis after histamine, and (3) that the patient not only had a 4 plus Wassermann and Kahn reaction but that the dermatologist considered her skin lesion as being due to syphilis which cleared up on antiluetic treatment.

In the next case of presumptive syphilis of the stomach the roentgenographic evidence was indistinguishable from that of carcinoma of the pylorus.

C. R. male, aged 37. The patient gave a 4 month history of epigastric pain occurring

during the course of a meal and at other times shortly before or after eating. The pain radiated around both sides to the back and often was associated with vomiting. He lost 29 pounds during this time. There was a history of a chancre 11 years previously for which he was treated.

Physical examination of the abdomen was essentially negative. There was a small eroded area on the prepuce.

Roentgenographic examination (Fig. 279 A) showed a persistent deformity of the prepyloric portion of the stomach which was diagnosed as carcinoma.

At operation. The region of the pylorus presented a circumferential thickening involving about 1 1/2 in. in the long axis. No crater was felt in this region but when the stomach was opened it was found that there was a shallow ulcer with serpiginous borders extending all the way around the lumen. The appearance of the ulcer was rather suggestive of a syphilitic process. There were moderately extensive adhesions between the posterior surface of the stomach and the posterior abdominal wall. These adhesions were unusually vascular. There were no enlarged nodes and nothing to suggest that the process was malignant.

A subtotal gastrectomy was done.

The pathologic examination of the specimen was reported by Dr. Douglas Symmers as follows:

Macroscopic examination. "Specimen consists of a portion of stomach including the pyloric end and about 15 cm. of duodenum. It measures about 10 cm. along the greater curvature. Inspection of the mucosa reveals it to be edematous and the rugae somewhat hypertrophied. Adjacent to the pylorus and apparently on the gastric side are two irregularly ulcerated areas with rolled edges and clean bases. They are about 1.5 cm. in diameter. The serosa overlying these areas appears to be unaffected."

Microscopic examination shows in the musculature numbers of small thin walled blood vessels with circumvascular round-cell infiltration. Other small vessels are hyalinized, thickened and completely or incompletely surrounded by a mantling of lymphocytes. Scattered throughout the muscle tissues are several microscopic collections of cells which suggest a possibility of milium gummatum. In one place is a relatively large collection of similar cells with a solitary giant cell of the Langhans type.

The lesion is a whole taken into consideration with the patient's history of a sore

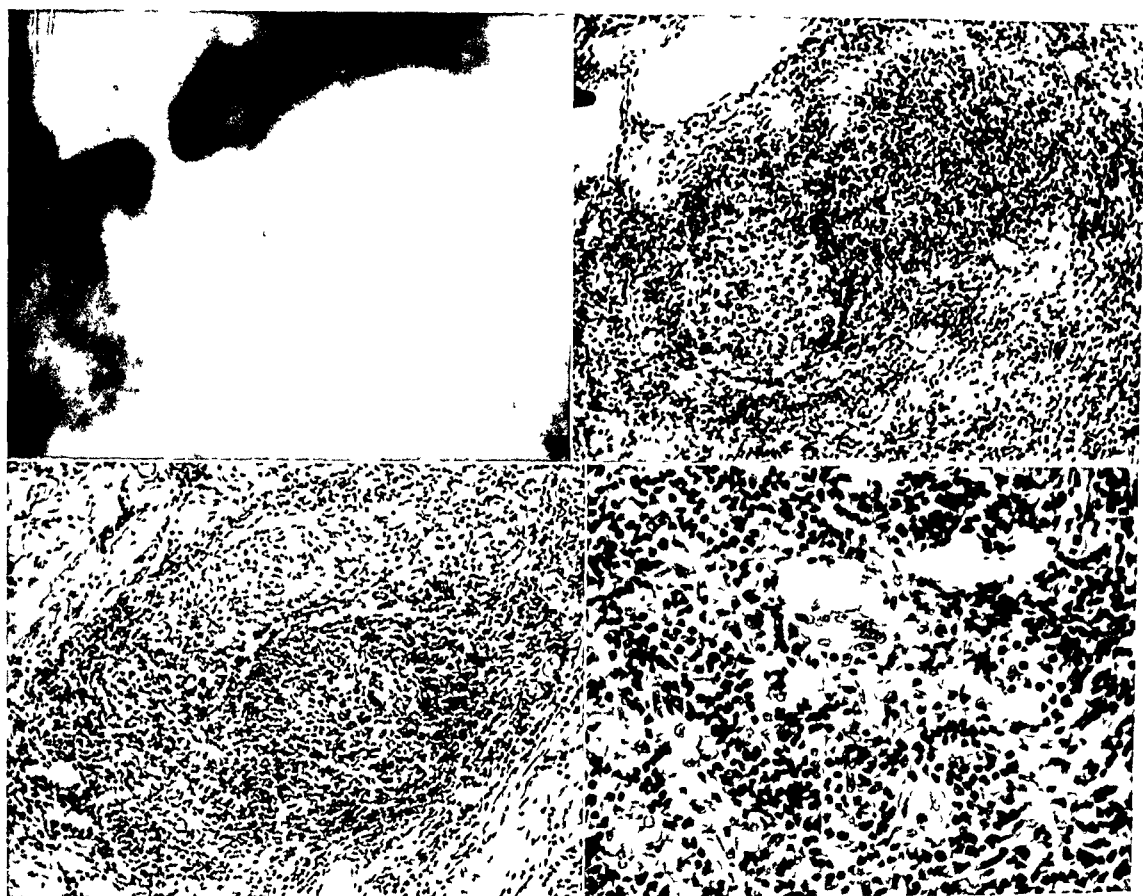


FIG 279 Syphilis of the stomach (A, *Top, left*) The deformity of the pylorus is indistinguishable from carcinoma (B, C, D) Photomicrographs showing the histologic characteristics of the lesion as described in the text

on the penis 11 years ago and the presence of a scar on the penis at the present moment, together with his statement that the Wassermann reaction was positive 11 years ago, combine to suggest the extremely rare lesion of syphilis of the stomach

"Histologic diagnosis Syphilis of stomach"
 Figures 279 B, C and D show the microscopic appearance of the lesion

Therefore, this is a reasonably well-confirmed case of syphilis of the stomach. From the roentgenographic appearance of the deformity produced by the lesion it is impossible to differentiate it from a malignant infiltration.

The deformity produced by tuberculosis of the stomach is shown in the following pathologically confirmed case.

B V, male, aged 53. The patient had been operated for a fistula-in-ano a year and a half

prior to his readmission to the hospital because of upper abdominal pain and vomiting. Vomiting began 6 months previously and occurred 3 or 4 times a week. The contents consisted of almost unchanged food without blood. Five months later he developed left-upper-quadrant pain, gnawing in character and occurring only when lying on his left side, it was relieved by a change in position. He lost 20 pounds during that period. Of possible clinical significance was the fact that 5 years previously he was treated for a tuberculous abscess of the third and the fourth lumbar vertebrae.

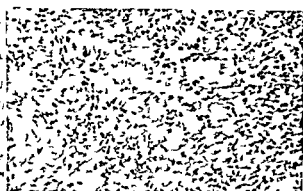
Physical examination revealed a well-developed and well-nourished white male in no acute distress. Firm, movable nontender axillary nodes were palpable, as well as a single small, firm, movable supraclavicular node. The abdomen was not tender. The liver was palpable 4 fingers' breadth below the right costal margin. There was possibly some fullness felt to the right of the umbilicus just



FIG 280 Tuberculosis of the stomach (A, *Top left*) Note the marked deformity of the pylorus with constriction of the lumen (B *Top right*) A detailed study of the deformity shows extensions of the barium to either side of the central area of constriction which may represent the sinus tracts found on pathologic examination of the resected specimen. This appearance may well have significance in the diagnosis of gastric tuberculosis (C *Center*) Low power Section reveals marked thickening of the submucosa by granulomatous reaction which extends into the muscularis and to a lesser extent into the lamina propria.



In this section the mucosa is intact (D, *Bottom left*) High power Section reveals details of the granulomatous reaction which is made up principally of epithelioid cells and occasional Langhans giant cells about which there is a peripheral rim of lymphocytes. There is necrosis in the center of this area (E *Bottom right*) Very high magnification Tuberculous granulation tissue consisting of epithelioid cells and Langerhans giant cells with peripheral lymphocytic infiltration.



below the liver edge Gastric analysis revealed no free acid after histamine injection

Roentgenographic examination (Fig 280 A) showed a deformity of the distal portion of the stomach with marked narrowing at the pyloric outlet A diagnosis of carcinoma of the stomach was made The detailed appearance of the constricted area may be noted in Figure 280 B In retrospect it is quite possible that the extensions to either side of the narrowed pylorus might have been produced by barium within fistulous tracts The diagnostic possibilities of this feature of the roentgen examination will be discussed at the end of the presentation The duodenal bulb was not clearly visualized at this time

At operation hard discrete nodules were found all along the lesser curvature of the stomach There were several nodules in the mesentery, as well as in the right and the left lobes of the liver A biopsy of the liver nodule and a node from the lesser curvature on frozen section showed caseation "typical of tuberculosis" The duodenum felt hard and firm, when opened, the lumen was found to be narrowed The pyloric mucosa was thickened and extremely friable A subtotal gastrectomy was done

Macroscopic examination of the resected portion of the stomach was reported as follows "The surface is injected the wall is thickened, firm and nodular The mucosa is injected, and at the pyloric end two shallow irregularly shaped ulcers are seen *On the mucosal surface numerous slitlike openings are present which lead to large pockets undermining the mucosa Some of these pockets communicate with small muco-pus-containing cavities in the deeper layers of the wall These changes extend to the line of resection*"

Microscopic examination "The stomach shows a gastric mucosa that gradually loses its orderly arrangement, becomes thin, infiltrated with lymphocytes and polymorphonuclear leukocytes and then becomes ulcerated The ulcer undermines the mucosa and is filled

with red blood cells and polymorphonuclears There is a gradual transition from this tissue to epithelioid granulation tissue that extends through the entire bowel wall so that the normal layers cannot be identified It is composed of epithelioid cells, lymphocytes, many plasma cells, as well as eosinophils and multinucleated giant cells There are numerous, small epithelioid granulomata that have necrotic centers, some of which contain polymorphonuclear cells and cellular debris Many giant cells are present in these granulomata In one of the sections an abscessed area with many polymorphonuclear leukocytes, red blood cells and cellular debris is seen just beneath the serosa

"Liver Section shows a small amount of liver tissue that rather abruptly blends into epithelioid granulation tissue and many multinucleated giant cells in this tissue which is similar to that seen in the stomach section

"Lymph node The architecture of the node is distorted by strands of epithelioid cells and discrete and confluent granulomata with small areas of caseation There are a few giant cells present Ziehl-Nielsen stain demonstrated occasional acid-fast bacilli in the lymph node Brown stains of all sections are essentially negative

"Diagnosis Tuberculosis of the stomach, lymph node and liver"

The microscopic appearance of the lesion is shown in Figure 280 C, D and E

In the light of the findings in the pathologic examination of the pylorus of slitlike openings which communicated with submucosal pockets, one may understand more clearly the roentgen appearance noted in Figure 280 B It is quite likely that the extensions above and below the area of major constriction may well be due to barium outlining these sinus tracts Such a roentgenographic deformity with evidence suggestive of sinus tract formations may be considered as roentgen evidence justifying the suspicion at least that the lesion is tuberculous in nature

REFERENCES

- 1 Brinton, W The Diseases of the Stomach, with an Introduction on Its Anatomy and Physiology, being lectures delivered at St Thomas Hospital, London, Churchill, 1859
- 2 Cornil, V Leçons sur la syphilis faites à l'hôpital de Lourcine, p 404, Paris, Baillière, 1879
- 3 Aschoff, L Pathologische Anatomie, ed 3, vol 2, pp 750, Jena, Fischer, 1913
- 4 Hartwell, John A Syphilis of the stomach, Ann Surg 81 767, 1925
- 5 Williams, C, and Kimmelstiel, P Syphilis of the stomach, JAMA 115 578, 1940
- 6 Eusterman, G B Gastric syphilis—ob-

- servations based on ninety three cases, JAMA 96 173, 1931
- 7 McNee J W Syphilis of the stomach, Quart J Med 15 215, 1922
- 8 Simmonds M Über Spirochaetenbefunde in Karzinomen München med Wchnschr 55 1103 1908
- 9 Luger, A, and Neuberger H Über Spirochaetenbefunde im Magensaft und deren diagnostische Bedeutung für das carcinoma ventriculi Ztschr klin Med 192 54 1921
- 10 Singer, H A and Dvas F G Syphilis of the stomach, with special reference to certain diagnostic criteria, Arch Int Med 42 718 1928
- 11 Harris S Jr and Morgan H J The isolation of *Spirochaeta pallida* from the lesion of gastric syphilis JAMA 99 1405, 1932
- 12 Fenwick W S A clinical lecture on syphilitic affections of the stomach Lancet 2 835 1901
- 13 Palmer, W I, Schindler R Templeton F E, and Humphreys E M Syphilis of the stomach a case report Ann Int Med 18 393, 1943
- 14 Beclere D and Ben aude R Un cas de syphilis gastrique—estomac biloculaire—trouble graves de la nutrition simulant un neoplasme—retour a la sante par le traitement specifique—controle radiologique Bull et mem soc med hop Paris 31 680 1911
- 15 Meyers J Syphilis of the stomach Albany Med Ann 33 563, 1912
- 16 Bockus H L and Bank J Upper gastro intestinal disease associated with syphilis JAMA 9 175 1928
- 17 Moore, A B and Aurelius J R Roentgenologic manifestations in eighty seven cases of gastric syphilis Am J Roentgenol 19 425 1928
- 18 Rokitansky, C A Manual of Pathologic Anatomy 2 43, Philadelphia, Blanchard & Lea, 1855
- 19 Coats, J On a case of tuberculosis of the stomach and one of acute miliary tuberculosis depending on tuberculosis of a pulmonary vein, with remarks on the pathology of these conditions, Glasgow M J 26 53 1886
- 20 Mus er, J H Tuberculous ulcer of the stomach Phila Hosp Rep 1 117 1890
- 21 Dewey K W Tuberculosis of the stomach with extensive tuberculous lymphangitis, J Infect Dis 12 236 1913
- 22 Broders A C Tuberculosis of the stomach with a report of a case of multiple tuberculous ulcers Surg, Gynec & Obst 25 490 1917
- 23 Pohl R Über Tuberkulose des Magens Rontgenpraxis 4 423 1932
- 24 Pop A and Hanganutin N Klinische und rontgenologische Betrachtungen über die Tuberkulose des Magens Zentralbl Chir 59 1629 1932
- 25 Renander, A Einige rontgenologisch beobachteten Fälle von Magentuberkulose Acta radiol 11 646 1930
- 26 Morris H R Gastric tuberculosis Am J Roentgenol 59 682 1948
- 27 Ostrum H W and Serber W Tuberculosis of the stomach and duodenum Am J Roentgenol 60 315 1948
- 28 Sherman R M and Johnston R L Tuberculosis of the stomach A case report Gastroenterology 16 593 1950

The Postoperative Stomach

ROENTGEN STUDIES FOLLOWING GASTRO-ENTEROSTOMY

The gastro-enteric stoma is circular or elliptical. For examination in cases involving it, only a small amount of barium should be used, since the stoma may be obscured if a large amount is employed. Also, large amounts may cause the greater curvature of the stomach to descend and overlap the gastro-enterostomy opening, thus making visualization difficult or impossible.

Roentgen examination may give valuable information about the stoma—its position, apparent size, evidence of functional derangement, irregularities of contour, fixation and tenderness. At the same time, it may disclose the nature of the original pathologic lesion for which operation was performed. Also of considerable importance is the mucosal appearance of both the stomach and the jejunum in so far as it can be determined roentgenologically. Abnormalities in the mucosal appearance, in the absence of any other evidence, may suggest the reason for the presence of abdominal symptoms.

In the study of the gastro-enteric stoma the following points are important. The first is the size of the stoma. It may be too large, so that the barium leaves with abnormal rapidity. The stomach may be empty in a few minutes. The jejunum may become markedly distended and filled in a homogeneous manner.

An abnormally rapid exit of the gastric contents through the stoma may lead to diarrhea and disagreeable sensations of fullness after meals, produced by the distention

of loops of small intestine from the rapid passage of the food. When the stomach contents leave too rapidly, there is insufficient time for hydrochloric acid—pepsin digestion. The mucous membrane of the jejunum becomes irritated because the food has not been sufficiently finely divided and because it enters the intestine in too large an amount. Moreover, the sudden dumping of the acid gastric secretion may disturb the normally alkaline jejunum. The acid coming from the stomach may partly inhibit the action of the pancreatic juice, which acts best in a neutral or slightly alkaline medium. The entire process of very rapid emptying through the gastrojejunal stoma, the dilatation of the coils of small intestine resulting therefrom, and the hypermotility of the small bowel may all be clearly visualized by fluoroscopic and roentgen examination.

On the other hand, the stoma may be narrowed, either because of the original technic of the operative procedure or because of secondary inflammatory change or spasm. The narrowing may be so marked that only a trickle of barium can be noted passing through the anastomosis. In some cases, no evidence of a functioning stoma is to be observed during a prolonged period of observation. Without any knowledge of the operative procedure, no report may be made regarding the absence of any evidence of a functioning stoma. This is one reason, among others, why a knowledge of the history and of the exact nature of any operative procedure is of great importance in any roentgen study.

The position of the stoma is important

When the stoma is placed too high on the greater curvature, there may be a disturbance in gastric evacuation. The stomach may empty well until the residue reaches below the level of the stoma. From then on, the stomach may have great difficulty in emptying itself, particularly if the pylorus is obstructed. Such a high position of the stoma may be due to an originally faulty technic, or the operation may have been performed on a markedly dilated stomach. The stoma originally well placed may have been carried to an abnormally high position with the gradual diminution in the size of the stomach.

The function of the stoma depends to some degree on its size. In addition, however, a rhythmic type of contraction has been described, simulating that of the activity of the pyloric ring and apparently vicariously taking over this function (Can non Kocher Case). However, to a large degree the opening of the stoma may be regulated by the contractions of the stomach. With relaxation of the stomach the stoma may remain open, and when the musculature of the stomach contracts, the stoma likewise may become narrowed. Thus, the emptying through the stoma may simulate that of an intrinsic sphincteric activity, although its function is secondary and really dependent upon the nature of the gastric tone.

Hellmer¹ described a rearrangement of the folds of the stomach and intestine at the site of anastomosis, so that the folds of the stomach and of the jejunum appeared to be heaped up at the stoma. He considered this arrangement as an expression of an intrinsic regulatory mechanism based on active movements of the mucosa according to the conception of Forsell. Such behavior according to Hellmer may thus be a factor in the production of a closure mechanism at the stoma explaining the sphincteric action occasionally noted in this region.

In some cases the emptying through the stoma is continuous without any evidence of a rhythmic function. In other cases the ejection of the barium occurs at irregular

times, there being no well defined relation ship between the periods of occlusion and the periods during which the stoma functions.

When no evidence of a functioning gastro enterostomy is demonstrable in a patient who has had such an operation performed, the explanation may be twofold. In the first place there may be an actual organic narrowing producing a stenosis. A poorly functioning or nonfunctioning stoma may also be evidence of edema and swelling of the mucosa and not necessarily of actual cicatrization. Spasm may be an important factor. Thus, a stoma that fails to function for a long period of time under fluoroscopic observation may suddenly permit the passage of barium and then continue to function in an apparently normal manner. Or having failed to show any evidence of function during the entire course of a single examination so that one is tempted to believe that either no gastro enteric stoma is present at all or that it is totally constricted, one may find, at a later re examination that a functioning stoma is actually present. Another possibility is that a stoma may show only a thin stream of barium making its exit from the stomach when the examination is conducted within a few weeks of the operation. That such narrowing can be due to temporary postoperative edema and swelling at the area of anastomosis is indicated by the fact that further study at a later period may exhibit an excellent stoma through which the barium leaves in an essentially normal manner.

In addition to a study of the size, position and function of the stoma, there are other points of importance in a study of this region. There may be fixation of the site of anastomosis. This is best determined by manipulation under fluoroscopic guidance.

As the result of the operative interference various organic abnormalities of the small intestine may be demonstrable. The vicious circle has been described whereby barium, entering the afferent instead of the efferent loop completely fills the afferent loop and extends back to the duodenum.

This condition is rarely ever seen at the present time, owing to improved surgical technic. In isolated instances, instead of a loop of jejunum being anastomosed to the stomach, a loop of ileum was mistakenly

substituted. I have personally examined a patient in whom an anastomotic communication was unfortunately established between the stomach and the transverse colon. The barium from the stomach promptly



FIG 281. (A *Top*) Narrowed stoma 15 days after gastroenterostomy (B *Bottom*) Three months after operation showing normal stoma



entered the large intestine, which was recognized by its characteristic haustral contour.

Postoperative adhesions following gastro enterostomy may be recognized particularly when they produce a partial obstruction or loops of small intestine which then show dilatation and stasis of the barium within them. At times, the folds of mucosa at the site of anastomosis may be abnormally prominent, and in some cases possibly may represent a jejunitis, in the absence of actual ulcer to account for the persistence of symptoms.

Inflammatory changes in the jejunum have been described following operation by gastroscopic observation (Henning, Schindler). Schindler described a case of chronic erosive jejunitis in a patient who had undergone a gastro enterostomy for duodenal ulcer. Gastroscopic investigation revealed irregular ulcerations at the margin of the stoma and for a short distance distally. The stomach showed an accompanying hypertrophic gastritis.

Schindler maintained that chronic gastritis is a frequent and serious complication of the postoperative stomach. Any of the various types of gastritis may occur. The

folds are swollen and edematous, erosions are often present and hypertrophic nodes may be noted. Massive hemorrhage is not uncommon. Schindler considered this type of gastritis to be secondary to the operative interference and stated that he examined gastroscopically, cases of duodenal ulcer prior to operation and found no evidence of gastritis only to demonstrate the presence of such gastritis at a later period after the operation. He blamed the development of the gastric changes upon abnormal function of the stoma with an unregulated reflux of intestinal contents, since he had never observed these changes in the presence of a rhythmically contracting stoma.

Illustrative Cases Narrowing of a postoperative gastro enteric stoma of an evanescent character and apparently due to temporary edema and spasm is illustrated by the following case.

J. T., male, aged 19. This patient had had recurring attacks of severe upper abdominal pain over a period of 2 years. This pain occurred 2 or 3 hours after meals. It radiated to the left flank and back. It would last for an hour or so and was frequently accompanied by vomiting. He noticed tarry stools.

The report after operation was as follows:



FIG. 282 High position of gastro enteric stoma

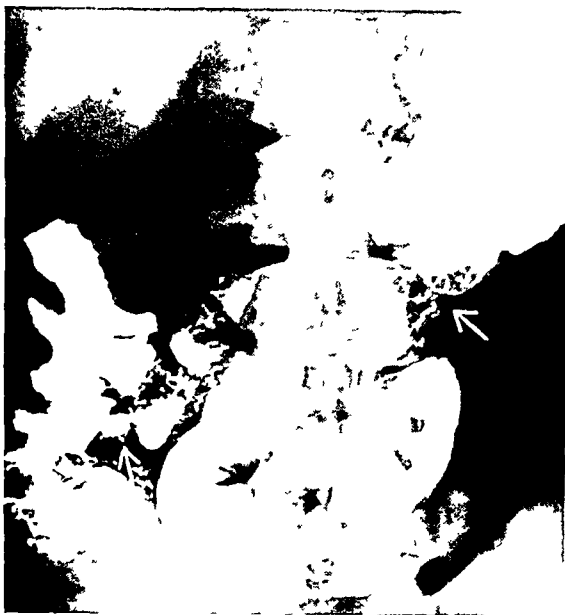


FIG 283 Gastro-ileostomy Note the short loop between the gastro-enterostomy and the ileocecal junction

"On the anterior superior surface of the first portion of the duodenum there was a thickened indurated area from which thick fibrous adhesions ran to the surface of the liver A gastro-enterostomy was done"

Roentgen examination about 15 days after the operation (Fig 281 A) showed marked narrowing at the site of anastomosis, with only a thin line of barium noted in this region, the barium then escaping into the afferent and efferent loops of jejunum Such a finding, if persistent, would indicate an organic constriction at the site of anastomosis

Roentgen examination 3 months after the operation (Fig 281 B) showed a stoma of normal lumen with excellent function, the barium passing fairly rapidly into the afferent and efferent loops of jejunum This change in the functional behavior of the stoma indicates that the narrowing present in the first examination must have been due to edema, inflammation and spasm still remaining from the original operative procedure That no actual organic constriction had occurred is proved by the normal nature of the gastro-enteric stoma as noted in the second examination 3 months after the operation The patient was symptomatically improved

Malposition of a gastro-enteric stoma is illustrated by the next case.

L K A duodenal ulcer was found at operation Gastro-enterostomy was performed Note the high position of the stoma as well as the marked dilatation of the proximal jejunum, apparently due to the initial rapid emptying of the stomach through the stoma (Fig 282)

Following the operation, the patient continued to have recurrent attacks of abdominal pain It is possible that the persistence of abdominal distress may have been due to the following factors

- 1 The persistence of the original duodenal ulcer At this time there is a marked deformity of the duodenal bulb

- 2 The marked dilatation of the proximal jejunum

- 3 The unusually high position of the stoma may interfere with the proper emptying of the distal region of the stomach

A rare and unfortunate complication is a gastro-ileostomy as illustrated by the following case

J H, male, aged 29 The patient, known to have pulmonary tuberculosis, had had a subtotal gastrectomy for duodenal ulcer at another institution 8 months before his admission to Bellevue Hospital From the time of the operation, he had had an almost constant diarrhea with from six to eight bowel movements every day, vague colicky abdominal pain and a weight loss of 55 pounds Physical examination of the abdomen was essentially negative except for a well-healed, midline abdominal scar

At operation it was found that the previous procedure had been of an anterior Polya type but that the stomach had been united to the ileum 2 feet from the ileocecal valve The anastomosis was taken down and a new one made between the stomach and the jejunum

Roentgen examination (Fig 283) showed that the stomach had been anastomosed to a loop of small intestine about 2 or 3 feet from the ileocecal junction The diagnosis was gastro-ileostomy

Another example of a gastro-ileostomy is shown in the case of A W, male, aged 52 The patient had a partial gastrectomy for a gastric ulcer 4 months previously Thereafter he began losing weight, and 2 months later developed an intractable diarrhea He noticed undigested food particles in the stool within 10 to 20 minutes after meals There was no pain, vomiting or blood in

the stool. He lost 40 pounds during that period.

Physical examination of the abdomen was essentially negative except for the scar of the previous operation.

Roentgenographic examination (Fig. 284) showed that the anastomosis of the remaining portion of the stomach to the small bowel was about 2 feet from the ileocecal junction. It was obvious from this examination that a distal loop of ileum had been anastomosed to the stomach. This was verified at operation and the gastroileotomy was dismantled. Examination of the resected stomach and ileum showed a tiny area of ulceration at the site of anastomosis. When the roentgenogram was reviewed it was noted that a very small cone-shaped niche was present at the medial border of the anastomosed ileum. This probably represented the small ulcer at that site found in the examination of the resected specimen.

The diagnosis of gastroileostomy may be made roentgenologically in two ways: (1) By the ingested meal method one may be able to show that there is only a short loop of small intestine between the gastroenteric stoma and the ileocecal junction. This observation may be confirmed if desired by (2) examination by means of a barium enema, showing the reflux of barium into the stomach by way of an abnormally short loop of small intestine.

The most important complication following gastroenterostomy is the development of a peptic ulcer of the jejunum. Such ulcers usually occur in the efferent loop

GASTROJEJUNAL AND JEJUNAL ULCER

The niche in the roentgenologic diagnosis of gastrojejunal or jejunal ulcer has the same value as in the diagnosis of peptic ulcer of the stomach or duodenum. Irregularities at the site of the anastomosis narrowing with pathologic gastric or gastrojejunal retention and tenderness localized



FIG. 284 Gastroileostomy. The anastomosis was about 2 feet from the ileocecal junction. Note the small cone-shaped niche of the ileum at the site of anastomosis apparently representing the ulcer found in the resected specimen.

at the site of the stomach are all suggestive and in combination may justify a diagnosis of ulcer in that region. When in addition however or even as an individual sign a typical niche can be demonstrated we have the criterion of greatest dependability for such a diagnosis. Here, too, this is due to the presence of the barium suspension within the crater. Where a gastroduodenal fistula has developed following gastroenterostomy, the presence of ulceration must be assumed as the cause.

Barsony² was apparently the first to demonstrate the roentgenographic appearance of a niche as evidence of gastrojejunal ulcer (1914).

Zollscher³ reported three cases in which he felt that a diagnosis of peptic ulcer of the jejunum was justified with operative proof in one case. In all three cases a gastroenterostomy had been performed with the development some time later of new symptoms accompanied by occult hemorrhages. On roentgenographic examination he found

a residual area of barium in the efferent jejunum painful to localized pressure. One of these patients was operated on, and the tender, nichelike area containing a barium residue was found to correspond to the crater of a jejunal ulcer. As the appearance in the other two nonoperated cases was similar, he believed that here, too, he was dealing with roentgen evidence of an ulcer niche.

Strom⁴ reported his experience following the examination of 300 patients after operation for ulcer and concluded that it was possible to demonstrate niche formation at the site of anastomosis as characteristic of ulcer, similar in diagnostic value to the finding of a niche in gastric or duodenal ulcer. Owing to the normally irregular appearance of the jejunum due to the valvulae conniventes, the demonstration of such niche formation may be much more difficult. This difficulty is increased if the involved area in the jejunum is overlapped by the barium-filled lower pole of the stomach.

In the technical demonstration of such niche formation, Strom emphasized the following points:

- 1 The importance of properly applied pressure to the area under observation
- 2 Giving the patient a second opaque meal several hours after the first, to demonstrate the relation of any nichelike area to the course of the gastrojejunal anastomosis
- 3 The value of several roentgenograms, to show the persistence of the defect

The significance of the niche in the roentgen diagnosis of jejunal ulcer soon received universal recognition.^{5, 6, 7}

Technical Aids in the Demonstration of the Niche. The demonstration of the niche is often difficult and requires detailed study of the anastomotic region. Mucosal studies with a small amount of barium and the use of compression are extremely important procedures in the study of this area. The objection to the employment of a large amount of barium is that the over-

filled greater curvature of the stomach may obscure the posteriorly placed lesion. On the other hand, there are cases in which the use of small amounts of barium may fail to fill out the niche of the ulcer, and overdistention may succeed in accomplishing this purpose. Therefore, a combination of both methods may be essential for the demonstration of a jejunal niche. Thus, in examining a patient for a suspected jejunal ulcer, the patient swallows a mouthful of the barium suspension. Under fluoroscopic guidance this is carefully spread over the area being studied. Films are taken, their number and the position of the patient depending upon the judgment of the examiner. Following this, the patient is then given enough barium to fill the stomach, and further observation is made with additional films, if required. Occasionally a flake of barium retained within the valvulae conniventes may simulate the appearance of a niche. However, careful observation will disclose the inconstant nature of this finding, or re-examination will fail to show the recurrence of this shadow at the identical region.

As in the case of a peptic ulcer elsewhere, at times one may be able to demonstrate increased prominence of the mucosal folds as well as radiation of these folds from the niche as a focal point. Also, one may be able to demonstrate in the erect position the presence of three layers: a lower layer of dense barium, an intermediary layer less dense in appearance, and a fluid level line surmounted by an air bubble.

As a result of cicatricial stenosis produced by the ulcer, there may be a prestenotic pouching produced by pulsion. These are analogous to the prestenotic pseudodiverticula which develop in the duodenal bulb as a result of the cicatrization of a duodenal ulcer. These pouches may be constant, remaining even after the healing of the ulcer itself.

In addition, just as in the case of the niche of a gastric or duodenal ulcer, occa-

sionally one may be able to show a shortening of the contour of the jejunum proximal and distal to the niche and also an incisura of the contour opposite the niche. In rare instances, mucosal studies of the anastomosed area may show a puckering of the gastric rugae so that they appear like the radiating spokes of a wheel converging upon the area of anastomosis where the jejunal ulcer is located.

Some of the postoperative irregularities of contour at the site of areas of anastomosis may be due to the actual process of suturing, with a turning in of the cut edges into the lumen.

In the presence of ulcer of the jejunum there may be a destruction of normal mucosal markings in this region with the presence of considerable narrowing. In fact, sometimes such evidence may be all that can be determined to indicate the presence of an ulcer in the absence of a niche. If the narrowing is marked it may lead to disturbance of gastric motor function and abnormal delay in the emptying of the gastric contents. Another coincidental finding in the examination of the stomach is the frequent evidence of unusual prominence of the rugal folds which in some cases may be the expression of a chronic gastritis.

Still another finding involving the stomach is the presence of an irregular concavity of the greater curvature, in the center of which the barium is seen emerging through the anastomosed jejunum.

Localized tenderness is an additional important aid in diagnosis. Finally, one may be able to demonstrate fixation, particularly by manual palpation during fluoroscopic examination.

Illustrative Cases. In the technic of examination the thin layer method is as important for the demonstration of a jejunal ulcer as it is in the case of a gastric or a duodenal ulcer. Thus Figure 285 A, with considerable barium present fails to show clear evidence of a jejunal ulcer. With a

thin layer of barium the niche of the jejunal ulcer is clearly seen (Fig. 285 B).

The importance of the thin layer technic in the demonstration of a postoperative jejunal ulcer is also illustrated in the following case.

N. L. male aged 72. The patient had been operated for "an ulcer" 14 years previously. He was quite well until 7 months before hospitalization when he developed severe epigastric pain radiating to the back and associated with vomiting. There was no history of hemorrhage from the gastrointestinal tract. The discomfort persisted in spite of medical management. He lost 9 pounds in the preceding 3 weeks.

Physical examination was essentially negative except for marked epigastric tenderness and the scar of the previous operative procedure.

Röntgenographic examination (Fig. 286 A) showed a gastroenterostomy. When the site of anastomosis was distended with barium there was no evidence of a niche. With a thin layer of barium a definite niche was clearly demonstrable just distal to the stoma (Fig. 286 B).

At operation the stoma was located at the middle portion of the stomach on the posterior wall at the greater curvature. The jejunum was densely adherent to the transverse colon and there was a penetrating ulcer at this point. The gastroenterostomy was disengaged, and a subtotal gastrectomy was done.

This case shows how readily the niche of a jejunal ulcer may escape visualization on cursory examination of the site of anastomosis when this region is overdilated with barium and the importance of the thin layer technic.

A characteristic example of postoperative jejunal ulcer is given below.

H. K., male aged 45. This patient had had periodic attacks of abdominal pain for 15 years. Nine years later he had a perforated ulcer which was repaired. Two years after that he had a recurrence of upper abdominal pain which resisted medical treatment. The following year he had a gastric operation. At the time of his admission to the hospital (3 years later) he complained of pain which was sharp occurring from 1 1/2 to 2 hours after

meals, and was relieved by food. He vomited frequently. The vomitus occasionally contained bile but no blood. There was no blood in the stools. He lost considerable weight.

Physical examination was essentially nega-

tive except for the evidence of the abdominal operations.

At reoperation the following report was made: "In the jejunum just distal to the gastro-enterostomy stoma, there was a crater



FIG 285 A (A, *Top*) Niche of a jejunal ulcer partly obscured by barium (B, *Bottom*) The jejunal niche is now clearly visible with a thin layer of barium





FIG 286 Postoperative jejunal ulcer (A, *Left*) The niche is not visualized at this time because the area of anastomosis is distended with barium (B, *Right*) With the thin layer technic the niche of the jejunal ulcer is visualized clearly

ulcer penetrating the wall of the jejunum densely adherent to the posterior wall of the transverse colon. Fixed in the wall of the ulcer were two pieces of linen suture, with their ends hanging loose in the crater. This was obviously the linen used in the original gastroenterostomy, and its presence in the wall of the ulcer seems very significant. The ulcerated area was hard, and there were fairly hard nodes in the mesentery. The stoma distal to it did not seem acutely inflamed, although there was some chronic induration of all the gastric wall and adjacent mucosa. A partial gastrectomy was done. The pathologic diagnosis was simple jejunal ulcer.

Roentgen examination (Fig 287) revealed the presence of a definite niche of a jejunal ulcer distal to the site of anastomosis. The contour to either side of the niche appeared retracted and shortened.

Another example of jejunal ulcer with shortening of the contour at the site of involvement is illustrated by the next case.

R. G. male, aged 39. The patient had had a gastroenterostomy for a duodenal ulcer 9 years previously. He remained comparatively well until 6 months before his admission to the hospital when he developed pain made worse by eating. He had a tarry stool on one occasion. There was no weight loss. Physical examination of the abdomen was essentially negative except for two well healed abdominal scars (one as a result of a previous appendectomy and the other the result of

the gastroenterostomy). At operation, in the distal jejunum, just beyond the gastroenterostomy stoma, an ulcer was found on the anterior wall. In the first portion of the duodenum, just beyond the pylorus, there was a cicatricial area, probably the site of the original duodenal ulcer. A partial gastrectomy was done.

Pathologic examination revealed marginal ulcer.

Roentgen examination (Fig 288) revealed

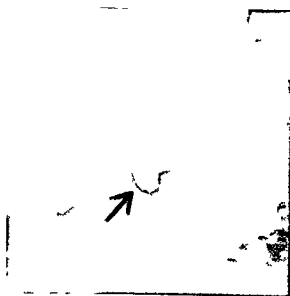


FIG 287 Niche of a jejunal ulcer with shortening of the contour of the jejunum to either side of the niche



FIG 288 Niche of a jejunal ulcer Shortening of the contour to either side of the niche



FIG 289 Niche of a jejunal ulcer Note the abnormality of the mucosal folds of the jejunum in the region of the ulcer



FIG 290 (Left) Large niche of a jejunal ulcer



FIG 291 (Right) Niche of a jejunal ulcer with incisura opposite

a gastro enterostomy and a niche originating in the efferent loop just distal to the site of anastomosis. There was shortening of the contour to either side of the niche. The mucosal folds in this region exhibited a definitely abnormal pattern. These were the findings upon which the diagnosis of postoperative jejunal ulcer was based. The essential criterion, of course, was the niche.

An abnormal appearance of the mucosal folds in relation to a jejunal ulcer is shown in the case that follows.

M. S., male, aged 67. The patient had a gastro enterostomy for duodenal ulcer and was symptom free for 1 year when he developed epigastric pain not relieved by food or medication. He vomited frequently. There was no blood in the vomitus. He had occasional tarry stools. Physical examination was essentially negative except for the operative scar and tenderness in the left upper quadrant. A subtotal gastrectomy was done. Pathologic examination revealed two marginal ulcers about 0.5 cm in diameter at the line of anastomosis. A third ulcer 0.5 cm in diameter was present just lateral to the stoma.

Roentgen examination (Fig 289) showed a niche at the site of anastomosis. The mucosal folds in that area were distorted. The preoperative diagnosis was jejunal ulcer. Although only one niche was clearly demonstra-

ble in the roentgenogram, there were three ulcers in the resected specimen.

Sometimes a jejunal ulcer may be very large.

P. C., male, aged 52. Five years previously the patient had had a gastro enterostomy for peptic ulcer at another hospital. He remained well for 1 year and then developed gastric pain, from 1 to 2 hours after meals, this pain was relieved by food and soda. On one occasion he was hospitalized for 3 weeks because of vomiting of blood.

Operation disclosed a marginal gastro jejunal ulcer penetrating into the pancreas and serosa of the transverse colon.

Roentgen examination (Fig 290) revealed a huge niche directly at the site of the gastro jejunal anastomosis. The diagnosis was jejunal ulcer.

Many of the phenomena in connection with the deformity of the niche of a gastric or duodenal ulcer may be noted in the case of a jejunal ulcer. In Figures 287 and 288, there was shortening of the curvature on either side of the niche. The following case of jejunal ulcer shows a niche with an incisura of the opposite side.

T. B., male, aged 36. This patient had a history of ulcer of 2 years' duration. He was oper-



FIG 292 Niche of a jejunal ulcer

ated on 1 year later. Following this he was symptom-free for about 3 months. From that time on he became incapacitated because of gastric pain. He passed tarry stools on several occasions. He showed no loss in weight. Physical examination was essentially negative.

At operation there were many adhesions around the gastro-enterostomy stoma and extensive veil-like adhesions between the upper coils of the jejunum and the lower surface of the mesocolon. When the anastomosis was exposed a marked thickening was found, particularly on the posterior surface of the afferent loops of jejunum. There was marked deformity and thickening of the transverse mesocolon, but the ulcer had not yet begun to penetrate toward the colon. A partial gastrectomy was performed.

Roentgen examination (Fig 291) made prior to operation revealed the following:

1. There was marked destruction of the normal mucosal markings of the jejunum just distal to the site of anastomosis.

2. Along the inner border was a definite triangular niche which was persistently present throughout the examination.

3. There was considerable localized tenderness at the site of anastomosis.

4. On the lateral border opposite the niche was a smooth incisura. The proximal loop of jejunum, about $1\frac{1}{2}$ inches long, appeared moderately dilated. This indicated the pres-

ence of partial obstruction at this site due to postoperative adhesions.

5. In addition, there was a retraction of the greater curvature of the stomach at the site of anastomosis.

As a postoperative jejunal ulcer progresses it may encroach upon the neighboring transverse colon. In some cases the inflammatory process may be limited to infiltration of the colon without actual ulceration and the production of a fistulous communication. In other cases, the process may go on to the development of an actual fistula. The following case is one in which there was infiltration invading the colon without ulceration into it.

J. Z., aged 62. This patient had undergone gastro-enterostomy for peptic ulcer. The persistence of symptoms eventually led to hospitalization and further surgical intervention. At that time the surgeon found a marginal ulcer at the site of the old stoma; in his opinion, this was confined to the gastric side of the stoma. Impending perforation was noted at this time. Infiltration had invaded the colon and the transverse mesocolon and its roots, so that these structures could not be separated. The indurated mass was about the size of a tennis ball and invaded the gastric wall about the stoma. This area of resection was solid.

Microscopic examination revealed no evidence of malignancy.

Roentgen examination prior to operation (Fig 292) revealed a cone-shaped niche just distal to the site of the previous anastomosis. Beyond the niche the jejunum was extremely narrowed, apparently the result of inflammatory induration.

OTHER COMPLICATIONS FOLLOWING GASTRO- ENTEROSTOMY

A remarkable complication of a gastro-enterostomy is related in the following case:

A. C. male, aged 66. The patient gave a history of having been operated on for a peptic ulcer 28 years previously. He was well until 2 months prior to admission to the hospital. He lost 15 pounds in those 2 months and during the preceding month had epigastric pain after meals relieved by vomiting. There

was no blood in the vomitus, but the stools were tarry during the preceding 2 weeks. Abdominal examination disclosed an old operative scar with a large incisional hernia and an orange size hard, slightly tender mass in the left upper quadrant.

Roentgenographic examination (Fig 293) showed the presence of a gastro enterostomy. There was narrowing and irregularity with obstruction just distal to the site of anastomosis. The transverse portion of the duodenum was partially obstructed. There was no other pathology of the stomach or duodenum. A diagnosis of an obstructive organic lesion at the stoma was made, the exact nature of which could not be determined.

Operation disclosed a fairly large mass at the site of anastomosis. There was no intrinsic lesion of the stomach. The tumor mass was resected.

The report based on the pathologic examination was papillary mucinous carcinoma arising at the margin of the gastrojejunostomy.

FIG 293 Deformity of the gastro enterostomy stoma due to a localized papillary mucinous carcinoma. (A, Left) Roentgenographic appearance of the deformed stoma. (B, Right top) Photomicrograph of the tumor. (C, Right bottom) High power magnification of the tumor.

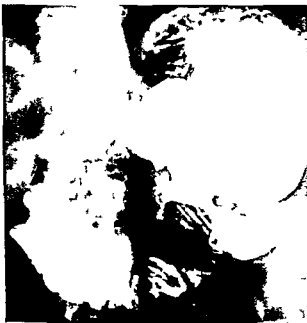


Figure 293 B shows a photomicrograph and Figure 293 C a high power magnification of the malignant tumor.

I have seen recurrent carcinoma of the stomach invade the site of gastrojejunostomy. However, this is my only experience with a case in which a carcinoma appeared to be limited to the gastrojejunostomy, although the stomach itself was otherwise entirely normal.

JEJUNOGASTRIC INTUSSUSCEPTION

Ledoux Lebard⁸ showed the roentgen appearance of invagination of the jejunum into the stomach secondary to gastro enterostomy. Within the stomach is to be noted the familiar pattern produced by the Krikling folds of the small intestine. A similar excellent demonstration of this process is to be found in the work of Lenarduzzi and Bonomini.⁹

Shackman¹⁰ in a review of the literature





FIG 292 Niche of a jejunal ulcer

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found 40 cases of jejuno gastric intussusception, to which he added one of his own. He found that the intussusception may involve either the afferent loop or the efferent loop and in some cases both loops may partake in the process.

McNamara's¹¹ case is unique in that the retrograde jejuno gastric intussusception occurred through the stoma of a subtotal gastrectomy.

How important the recognition of a jejuno gastric intussusception may be is illustrated by the severity of the symptomatology in the case reported by Lawson and Whitener¹² in which autopsy disclosed that it had produced an acute obstruction of the intussuscepted loop of jejunum within the stomach. At the site of the former gastro-enterostomy both loops of jejunum were dilated and disappeared into the stomach. The stomach contained, in addition to 3,300 cc of thin brownish fluid, a small sausage-shaped tumor arising out of the stoma of the gastrojejunostomy. This mass was the efferent loop of jejunum which had become invaginated in a retrograde manner through the stoma of the gastro-enterostomy. The stomach contained over 20 cm of gangrenous doubled jejunum. There was complete obstruction at the point of intussusception, and the rest of the small intestine and the large intestine was collapsed. A similar condition of retrograde intussusception of jejunum through the stoma after a subtotal gastrectomy was described by Grimes¹³. At autopsy the remaining portion of the stomach was considerably increased in size, and a soft mass could be palpated within its lumen. On opening the stomach it was noted that a retrograde intussusception had occurred of the efferent portion of the jejunum through the gastrectomy stoma. The distal end of the jejunum was necrotic and dark in color.

A fully confirmed case of jejuno gastric intussusception with excellent roentgenographic evidence may be found in the report by Aleman¹⁴. One may note the presence of

dilated jejunum, characterized by the valvulae conniventes, within the confines of the stomach. At operation about 50 cm of the efferent loop of the jejunum was found invaginated within the stomach through the gastro-enterostomy stoma. The operation was performed one hour after the roentgenographic recognition of the condition, and reduction was accomplished readily by withdrawing the intussuscepted loop.

PERFORATION AND FISTULAE

One of the earliest descriptions of perforation of a postoperative jejunal ulcer was that by Braun in 1899.¹⁵ The jejunal ulcer had developed in the efferent loop 11 months after gastro-enterostomy. Autopsy revealed a punched-out ulcer of the jejunum 1 cm distal to the stoma.

In addition to perforation into the free abdominal cavity, a jejunal ulcer may break through into the colon with the production of a gastrojejuno colic fistula.

In the demonstration of a gastrocolic fistula secondary to the perforation of a jejunal ulcer into the colon, roentgen examination may be carried out either by the ingested-meal method or after the administration of a barium enema. Not uncommonly, when barium is given by mouth in such cases, it may be impossible to demonstrate the area of fistulous communication. If a barium enema is administered, the reflux of the barium into the stomach will be clearly noted. It is for this reason that in the case of a patient who at some time after a gastro-enterostomy develops diarrhea, foul gaseous eructations and marked loss of weight and in whom a gastrocolic fistula is suspected, the roentgen study should not be considered as complete unless a barium enema has also been included. A patient should not be dismissed with the assumption that no gastrocolic fistula exists simply because this was not demonstrable after the administration of the barium suspension by mouth.

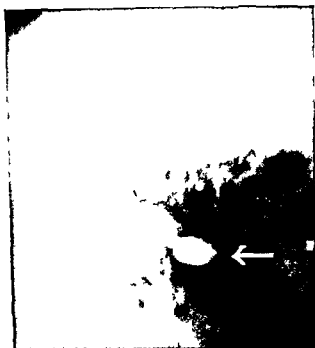


FIG 294 (*Left*) Niche of a jejunal ulcer

FIG 295 (*Right*) Gastrocolic fistula When the barium injected by enema reached the splenic flexure it entered the stomach



In addition to the actual fistulous communication between stomach and colon, roentgen examination may also disclose the niche itself, thereby giving a definite clue in addition to that furnished by the history as to the etiologic factor underlying the development of the fistula

As the barium administered by way of the colon enters the stomach the loops of jejunum leading from the area of anastomosis also become filled. If too much barium is introduced by enema, there may be such an overlapping of colon, stomach and numerous loops of jejunum as to make it difficult or impossible to localize clearly the exact area of communication between stomach and colon. Therefore it is preferable that the barium suspension be introduced slowly and as soon as some is noted entering the stomach that the examination be terminated promptly and roentgen exposures made.

While a combination of the ingested meal and the barium enema method of examination will disclose the existence of a gastrocolic fistula, failure to demonstrate its presence roentgenologically does not rule out the possibility that a small fistula is present. The opening may be partially occluded by retained fecal material, inflammatory edema, or the overlapping of mucosal folds in such manner as to make the entrance of the barium impossible at the particular time of examination.

Illustrative Cases Actual perforation of a jejunal ulcer into the colon may occur and yet in some cases it may be impossible to demonstrate the fistula communicating with the colon by the ingested meal method even though the niche of the jejunal ulcer can be noted. This is illustrated by the following case.

C. L., aged 51. For about a year this patient had had epigastric distress occurring about

2 hours after eating, this pain radiated to the back. He also had tarry stools. A gastro-enterostomy was done for duodenal ulcer. The patient then remained well for about 13 years when he developed weakness, loss of weight, and nonradiating epigastric pain, which occurred about 2 hours after meals and was relieved by soda. He vomited on one or two occasions. There was no blood in the vomitus or in his stools. He complained of diarrhea. Physical examination of the abdomen was negative.

At operation both limbs of the jejunum proximal and distal to the anastomosis were dilated to about three times their normal diameter. There were dense adhesions between the transverse colon and the jejunum, which extended from the site of the gastro-enterostomy along the distal loop for 4 inches. In this mass there was an opening between the jejunum and the colon. The original duodenal ulcer appeared to be healed. After the separation of adhesions between the jejunum and the colon, a fistulous tract was found at this point. The finger could be introduced through the opening into the transverse colon and the jejunum and through the gastro-enterostomy opening into the stomach.

Roentgen examination (Fig 294) revealed definite evidence of the niche of a jejunal ulcer. However there was no evidence of any gastrojejunal fistula such as was found at operation. This failure of demonstration of the fistulous tract may be due to one of two reasons. (1) It is not unusual, even when a gastrocolic fistula is present, not to be able to demonstrate it by the ingested-meal method, whereas examination following the administration of a barium enema may succeed in showing the entrance of barium from the colon into the stomach and jejunum. (2) Since the operative intervention was not done until 14 days after the completion of the roentgen examination, it is possible that the fistulous tract may not have been present at that time but developed later during this interval of 2 weeks.

The appearance of a gastrocolic fistula is well illustrated by the next case.

A B, aged 49. This patient had undergone gastro-enterostomy for peptic ulcer. He remained well for 7 years. Eight months before his admission to the hospital he developed diarrhea and fecal vomiting. He had lost about 30 pounds in the preceding year. On one occasion he collapsed suddenly on the street after

vomiting dark-brown material. Blood was present in the stool.

The report at operation was as follows: "There was a two-finger-breath gastro-enterostomy opening near the cardiac end of the stomach, about $\frac{3}{4}$ inch distal to which was a fistulous opening admitting one finger between the jejunum and transverse colon. There were dense adhesions of omentum to the peritoneum at the site of the incision and dense adhesions around the jejunal end of the gastro-enterostomy opening. The fistulous communication was repaired."

Roentgen examination prior to operation (Fig 295) revealed the following: After the administration of a barium enema, the colon filled up to the splenic flexure. As the barium passed on to the left half of the transverse colon, some of the barium was noted entering the stomach. Barium was also noted escaping into the coils of jejunum.

The diagnosis was gastrocolic fistula. Previous examination by the ingested-meal method failed to show the existence of the fistulous communication.

An example of gastrocolic fistula with visualization of the niche of the jejunal ulcer responsible for this eventual development is shown in the next case.

A M, male, aged 60 years. This patient had undergone gastrojejunostomy elsewhere. Two years later he was admitted to the hospital for gastric hemorrhage.

The report at operation was as follows: "On the posterior surface of the stomach at the site of the gastrojejunostomy, the transverse colon was adherent. Apparently there was a large, indurated ulcer involving all three structures and causing obstruction to the outlet of the stomach and also obstruction to the transverse colon. The stomach was markedly distended."

The autopsy report stated: "The stomach is markedly distended. The jejunum is attached to the middle of the greater curvature just anterior to the inferior border of the stomach, the stoma admitting two fingers. The stomach contains a fecal-like fluid having a fecal odor. The mucosa is congested. On the greater curvature, about $1\frac{1}{2}$ inches from the pyloric antrum, is a small opening admitting the tip of the index finger. The opening extends downward to the right of the jejunum. On the medial wall of this tract is a marginal ulcer about the size of a fifty-cent piece,

at the site of the anastomosis. The barium then entered a segment of bowel which, because of the haustral configuration, was obviously colon.

The diagnosis was gastrocolic fistula.

Operation revealed a gastrojejunocolic fistula. A jejunectomy, a partial colectomy (transverse) and a subtotal gastrectomy were done. The patient did very well. A follow-up 2 months and 11 days later showed that the patient had gained 46 pounds.

HEALING OF JEJUNAL ULCER

Similar to the disappearance of the niche of a gastric or a duodenal ulcer under medical management, the niche of a jejunal ulcer may also show complete disappearance. The mere finding of a jejunal ulcer, therefore, does not justify prompt surgical intervention. A period of medical treatment is justified in jejunal ulcer as in gastric or

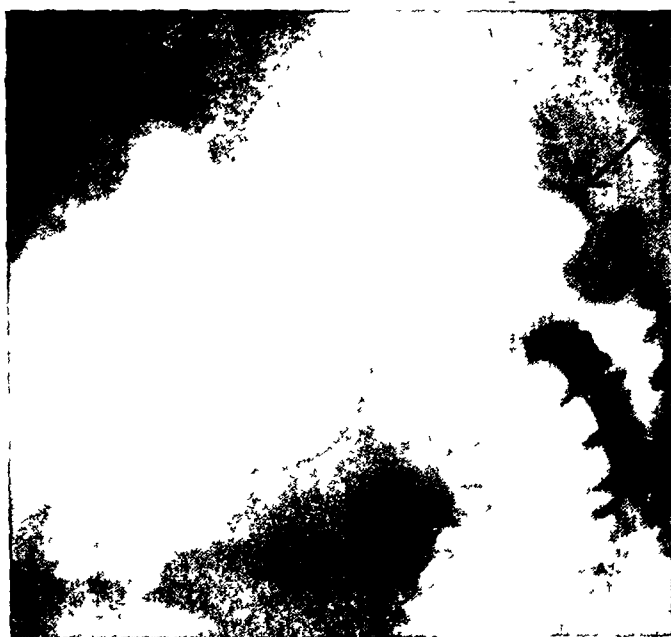


FIG 298 (A, *Top*) Niche of a jejunal ulcer (B, *Bottom*) Note almost complete disappearance of the niche after medical treatment

duodenal ulcer. The niche of a jejunal ulcer may also show an increase in size in the roentgenogram.

That the niche of a jejunal ulcer may exhibit roentgenologic evidence of complete disappearance under medical management similar to the niche of a gastric or duodenal ulcer was shown by me in 1934.¹⁶

Marked diminution in the size of the niche of a jejunal ulcer can be noted in Figure 298 A and B. Figure 298 A shows

the original appearance of the niche. Figure 298 B shows considerable regression in the size of the niche after medical treatment.

Not only may the niche of a jejunal ulcer show a diminution in size but, rarely, one may trace an increase in the size of the niche. This transformation is illustrated by the next case.

J. M., aged 60. About a year after gastroenterostomy for postpyloric ulcer, this patient was admitted to the hospital with a his

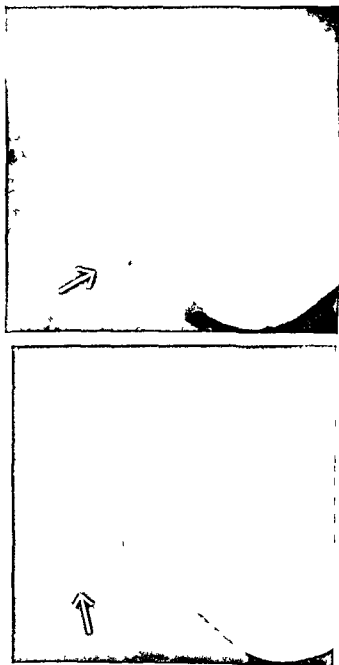


FIG. 299 (A, Top) Niche of a jejunal ulcer. (B, Bottom) Note the marked increase in the size of the niche 6 months later.

tory of epigastric distress (gradually becoming more severe), frequent attacks of hemorrhage and a loss of 30 pounds in weight

The roentgen examination is illustrated in Figure 299 A and B. Figure 299 A shows the appearance of the jejunal niche at the first examination. An examination about 6 months later Figure 299 B showed the jejunal niche considerably increased in size

This patient was later operated on and was also examined at autopsy. The jejunal ulcer was found. In addition, there was a gastrocolic fistula, which had been demonstrated roentgenologically

ROENTGEN FINDINGS AFTER OTHER OPERATIVE PROCEDURES

Roentgen examination may also be carried out following other operative procedures, such as pyloroplasty, closure of an acute perforation of an ulcer, V-shaped resection and partial gastrectomy

Pyloroplasty The findings on roentgen examination after pyloroplasty may be of a marked irregularity of contour at the pyloroduodenal region of such nature as to

simulate the deformity of an actual ulcer. Differentiation may be practically impossible except on the basis of the history. Nor can one determine reactivation in the region of operation unless a niche is shown.

Closure of a Perforated Ulcer A frequent postoperative examination is that following the simple closure of a perforated ulcer. In some cases of perforated gastric ulcer, there may be a recurrence of a niche at or near the site of operation. A persistent incisura may develop on the greater curvature opposite

Persistence of a gastric ulcer at the original site of acute perforation is shown by the following case

G. S., male, aged 36. This patient gave a 3-week history of epigastric pain occurring from 1 to 2 hours after meals. He had a sudden attack of acute, upper-abdominal pain. At operation, a perforated gastric ulcer was found, about 4 mm in diameter, and the induration extended from the perforation for a distance of 3 cm in all directions. Turbid fluid was present in the peritoneal cavity. Because of the marked induration surrounding the ulcer, closure was difficult but it was finally



FIG. 300 Recurrence of a gastric niche after suture of the perforation



FIG 301 Subtotal gastrectomy with normal stoma all of the barium escaping from the stomach by way of the efferent loop



FIG 302 A Subtotal gastrectomy Note the escape of the barium by way of the afferent and the efferent loops

FIG 302 B Note the normal mucosal pattern of the anastomosed jejunum There is no abnormal distention of the loops nor any evidence of deformity of structure



accomplished by one layer of mattress sutures. A portion of the omentum was brought up and sutured at the site of the perforation as well.

Roentgen examination 2 weeks later showed the presence of a large niche of the lesser curvature indicating that the operation had failed to eradicate the ulcer (Fig. 300).

V shaped Resections Local excisions may be in the form of V shaped resections at either the greater or the lesser curvatures. In the case of the greater curvature a change in the form of the stomach may not be demonstrable. A V shaped excision at the lesser curvature may produce an hourglass appearance as a result of the contraction of the circular muscle fibers and simulate the appearance of hourglass formation secondary to ulcer. There may be recurrence of ulcer at the site of a localized resection. The lesser curvature may appear shortened similar to the shortening pro-

tory of epigastric distress (gradually becoming more severe), frequent attacks of hemorrhage and a loss of 30 pounds in weight

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FIG 300 Recurrence of a gastric niche after suture of the perforation



FIG 301 Subtotal gastrectomy with normal stoma all of the barium escaping from the stomach by way of the afferent loop

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FIG 302 A Subtotal gastrectomy. Note the escape of the barium by way of the afferent and the efferent loops.

FIG 302 B Note the normal mucosal pattern of the anastomosed jejunum. There is no abnormal distention of the loops nor any evidence of deformity of structure.





FIG 303 (*Left*) Subtotal gastrectomy. Note the unusual prominence and tortuosity of the mucosal "relief" pattern of the stomach.

FIG 304 (*Right*) Partial gastrectomy with abnormally rapid emptying through the stoma.

duced by an ulcer associated with considerable scar formation.

In the presence of a history of a gastric operation of doubtful nature, the finding of a persistent incisura of the greater curvature may lead to the suspicion that the original surgical procedure had been a V-shaped resection of an ulcer of the lesser curvature opposite.

In some cases there may be no roentgenologic vestige of the original operative procedure. This is particularly true when a benign tumor is submucosal and has been enucleated without any resection of the gastric wall. In those cases in which a portion of the wall of the stomach to which the tumor was attached has been removed, the curvature may appear shortened.

Gastric Resection. Gastric resection may be done for peptic ulcer or for gastric tumor. Examination includes a careful

study, not only of the region of the anastomosis, but of the remaining stomach as well.

Figure 301 shows a subtotal gastrectomy with all of the barium leaving by way of the efferent loop. The stoma functions well, the barium suspension from the stomach passing through it with normal rapidity. The mucosal pattern of the jejunum at the site of anastomosis is normal.

Figure 302 A shows a normally functioning subtotal gastrectomy with the barium emerging by way of both the afferent and the efferent loops.

The normal mucosal pattern of the anastomosed jejunum in subtotal gastrectomy is shown in Figure 302 B.

In the subtotal gastrectomy illustrated by Figure 303, the mucosal folds of the stomach are unusually prominent and tortuous. This appearance of the gastric mucosa may

antecede the operative procedure. In some cases, however, this abnormality of the gastric mucosal relief is an aftermath and may be due to a postoperative gastritis.

As in the case of simple gastro enterostomy, the size of the stoma is of importance in preventing too rapid emptying of the stomach with secondary overdistention of the small intestine.

Figure 304 illustrates one of the important complications following partial gastrectomy when the stoma is too large and the gastric contents are hurried into the small intestine with abnormal rapidity. The intestine becomes overdistended and this may lead to serious abdominal disturbances.

Following anastomosis of the jejunum to the stomach there may be inflammatory changes at the site of operation. The rugal folds of the stomach may show unusual prominence and tortuosity. The wall of the small intestine may show diminished flexibility. Mucosal folds in the jejunum may be unusually prominent and may be obliterated with difficulty or not at all. The contour of the jejunum may be irregular, and the stoma narrowed. The normal jejunal folds may be effaced. The same procedures previously outlined for the study of the stoma following gastro enterostomy are applied to the study after partial gastrectomy.

A postoperative jejunal ulcer may be an important complication of partial gastrectomy. The following case illustrates an occurrence of this nature.

B S, male, aged 44. Thirteen years after gastro enterostomy for gastric ulcer, this patient had an emergency operation for repair of a perforated marginal ulcer. Three years later he had a gastric resection because of bleeding jejunal ulcer. He then remained symptom free for a while. He then began to have crampy pains which continued for 7 months up to the time of his admission to the hospital. The pain was relieved by soda. He vomited on many occasions. Physical examination was negative. He had lost considerable weight.

Upon operation a large jejunal ulcer with a marked induration around the jejunum was



FIG 305 Jejunal ulcer after partial gastrectomy

found at the site of the gastro enterostomy stoma. The jejunum was dissociated from the stomach. Part of the remaining stomach was resected.

Roentgen examination prior to this last operation (Fig 305) showed just distal to the site of anastomosis a large niche characteristic of ulcer. This was corroborated at operation, as reported above.

Recurrences of jejunal ulceration after partial gastrectomy in spite of repeated surgical intervention is illustrated by the following case. In addition, a gastrojejuno colic fistula had developed.

A B, male, aged 36. For 5 years the patient had had recurrent attacks of upper abdominal pain, which were worse at night. He then had an acute perforation of a duodenal ulcer which was promptly sutured. Nine months later he had a recurrence of symptoms. Roentgen examination showed an ulcer of the duodenum and a subtotal gastrectomy was done. Three months after this operation there was a return of symptoms and roentgen examination showed a jejunal ulcer. He was reoperated and the jejunal ulcer resected. From then on his symptoms were worse than they had ever been and further roentgenographic examination disclosed the fact that a jejunal ulcer was present (Fig 306 A). (Note

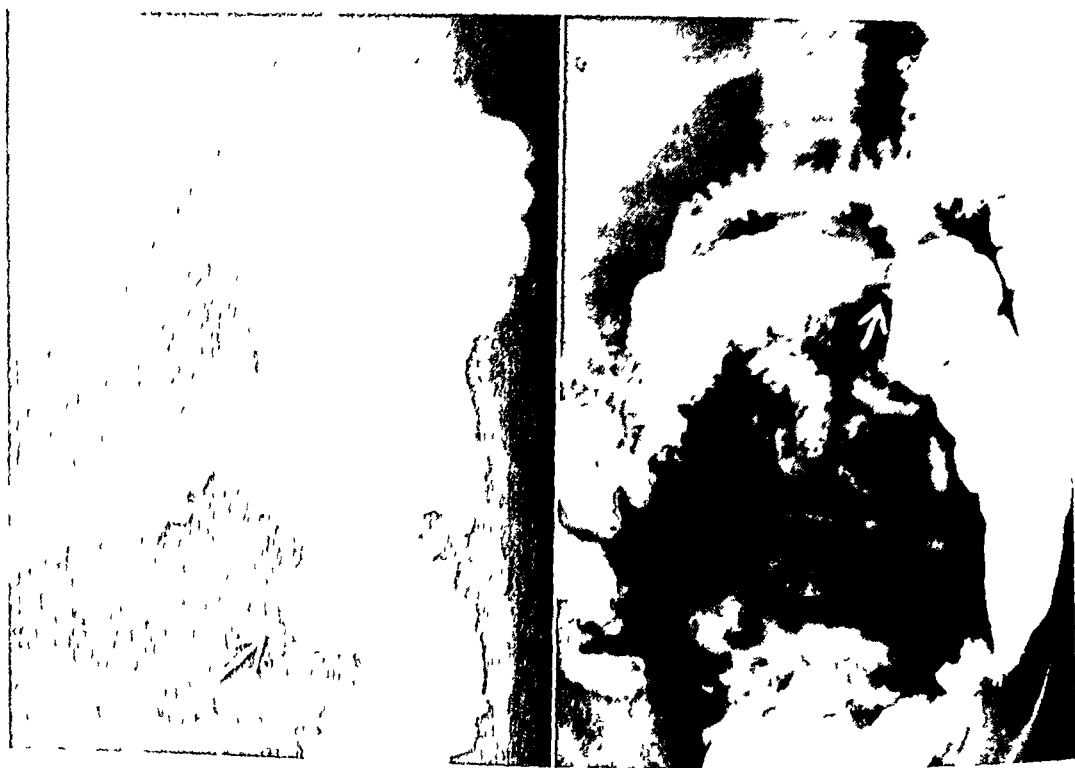


FIG. 306 (A, *Left*) Jejunal ulcer after partial gastrectomy (B, *Right*) Same patient. Gastrocolic fistula. Note reflux of barium into the stomach following a barium enema

the large niche at the site of anastomosis.) In addition, there was a gastrojejunal fistula, demonstrated by means of a barium enema with reflux into the stomach (Fig. 306 B).

These findings were verified at a subsequent operation. The gastrocolic fistula was closed off, and further resection of the jejunal ulcer was done. The patient remained well for about 4 months, when all his symptoms re-



FIG. 307 (*Left*) Jejunal ulcer after partial gastrectomy
FIG. 308 (*Right*) Jejunal ulcer after partial gastrectomy. Note the marked distortion of the mucosal pattern at the site of anastomosis.

turned and at the last examination, unfortunately, there was roentgen evidence of another jejunal ulcer

The next case is that of a brother of the preceding patient

N B, male, aged 41 The patient gave a 20 year ulcer history Three months before his admission to the hospital he had had a recurrence of pain which resisted medical management The pain was in the epigastric region, burning in character and relieved for only $\frac{1}{2}$ hour by milk or alkaline powder or aluminum hydroxide

Physical examination of the abdomen was essentially negative A partial gastrectomy was done for a duodenal ulcer About $1\frac{1}{2}$ years later the patient had a recurrence of symptoms with pain relieved by food and alkaline powders He was operated upon again and a jejunal ulcer was found

Roentgen examination (Fig 307) prior to the last operation showed a large niche in the efferent loop of jejunum just distal to the site of anastomosis The diagnosis was postoperative jejunal ulcer

Note the similarity in the appearance of the niche to that in the case of his brother A B (Fig 306 A)

In the next case a jejunal ulcer recurred in spite of surgical removal with more extensive gastric resection than had been done in the original partial gastrectomy

T C, aged 37 The patient gave a classical ulcer history, for which a partial gastrectomy was done at another institution Soon thereafter he developed excruciating pain, mainly in the left upper quadrant, which frequently kept him awake all night The pain was somewhat relieved by milk and aluminum hydroxide He responded for a while to medical management only to have a recurrence of severe intractable pain At operation a penetrating jejunal ulcer was found at the margin of his gastrojejunostomy stoma

After freeing the penetrating marginal ulcer the gastrojejunostomy was dismantled and approximately 2 inches more of the stomach was resected The patient's condition clinically was not improved and he later developed another jejunal ulcer in spite of the more extensive gastric resection

Figure 308 shows the niche of the jejunal ulcer in the examination made after his first operation



FIG 309 Niche of jejunal ulcer involving the afferent loop of the jejunum The constriction of the lumen of the proximal portion of both the afferent and efferent loops was the result of inflammation and edema of the wall

An ulcer may also develop on the afferent loop of jejunum after subtotal gastrectomy

The patient (V M, male, aged 62) gave an ulcer history of almost 25 years duration and a subtotal gastrectomy with gastrojejunostomy was performed 8 years prior to the present hospitalization Intermittent attacks of epigastric pain persisted for which he received medical treatment The patient's distress became worse and on one occasion was accompanied by vomiting Two days before admission to the hospital he noted tarry stools which persisted up to the time he entered the hospital

Physical examination of the abdomen was essentially negative except for a well healed upper midline scar and slight epigastric tenderness

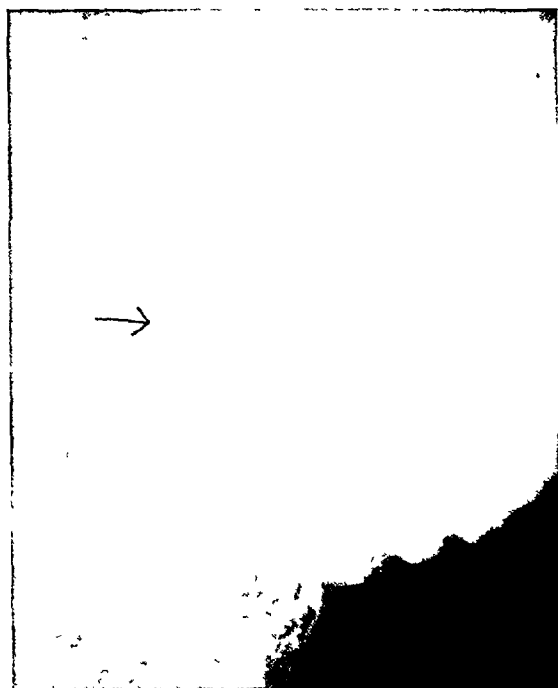


FIG 310 Recurrence of gastric carcinoma after partial gastrectomy

deanness While under observation he vomited 500 cc of bright red blood The patient received whole blood transfusions and recovered from the hemorrhage

Roentgenographic examination (Fig 309) had disclosed previously a subtotal gastrectomy with a niche involving the afferent loop of the anastomosed jejunum just distal to the stoma The jejunum to either side of the niche was constricted, and the rest of the afferent loop was quite dilated The proximal segment of the efferent loop for a distance of about 1 inch beyond the site of anastomosis was narrowed and irregular in configuration The preoperative diagnosis was jejunal ulcer with inflammatory pathology of the proximal portion of both the afferent and the efferent loops of jejunum

At operation "There was a marginal ulcer of the afferent limb of a previous anterior Polya subtotal gastrectomy The ulcer was near the line of anastomosis with the stomach It appeared to be quite indurated with an associated area of edema of the afferent and the efferent limbs of the jejunum The jejunal walls were boggy and edematous, as was its mesentery The stomach appeared to be approximately resected for a distance of $\frac{2}{3}$ in A revision of the gastrectomy was done with resection of the jejunal ulcer"

Pathologic diagnosis Chronic ulcer at the

site of anastomosis between the stomach and the small intestine

In this case the preoperative roentgen diagnosis of a jejunal ulcer of the afferent loop was confirmed operatively The narrowing of the lumen of the anastomosed loops of jejunum was evidently the result of induration and edema of the walls

Of particular importance following resection for malignancy is the question of recurrence of the malignant lesion It is possible, of course, for metastatic invasion to take place at a distance from the original resection and be roentgenologically unrecognizable In addition, evidence of recurrence may be noted in the remaining portion of the stomach and may be indicated roentgenologically by an irregular configuration Such evidence must be carefully differentiated, however, from irregularities at the site of anastomosis resulting from the operative procedure itself, as well as from unusually prominent and tortuous gastric folds When the irregularity is due to a recurrence of the malignancy, the deformity can be noted well beyond the confines of the zone of operation and extending high up toward the cardia A mucosal study demonstrating the rugal folds as well as the moth-eaten irregular nature of the gastric contour will aid in the differentiation of actual organic infiltration from the intact mucosal structure Palpation of the tumor may show that it coincides with the irregular gastric contour There is apt to be diminution of mobility of the stomach due to fixation of the mass to neighboring structures

Recurrence of new growth in the part of the stomach still remaining after partial gastrectomy is illustrated by the following case

O G, male, aged 61 For about 2 years this patient noticed the gradual onset of vomiting and of occasional epigastric pain not related to meals The pain was boring and did not radiate, it was relieved to a considerable degree by vomiting Gastric resection was performed for an adenocarcinoma of the pyloric portion of the stomach

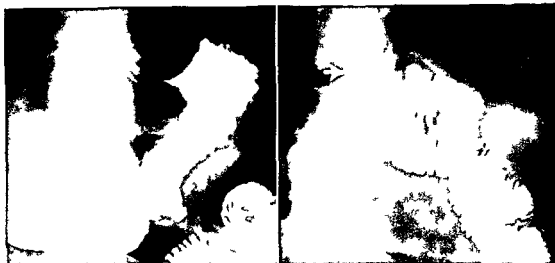


FIG 311 (A, *Left*) Carcinoma of stomach prior to total gastrectomy (B, *Right*) Esophagojejunostomy after total gastrectomy

The patient remained symptom free for almost 2 years, when he began again to experience epigastric pain, relieved by vomiting. The vomitus often contained partially digested food eaten from 2 to 3 days previously. He had lost about 60 pounds in the preceding 2 years.

Physical examination of the abdomen revealed a mass in the epigastrium extending from the substernal region to the umbilicus. It was tender on pressure.

The report at operation was as follows: "The entire peritoneal lining of the supracolic compartment of the abdomen, both visceral and parietal, was studded with milium-like nodules. A huge carcinomatous mass invaded the transverse mesocolon obstructing the gastroenterostomy stoma and extended on to the distal portion of the remaining stomach. The liver showed no palpable gross metastatic nodules. On palpation there appeared to be some evidence of a gastrointestinal stoma although this was encroached upon in all directions by the malignant mass. The condition was considered inoperable. Radon seeds were implanted."

Roentgen examination (Fig 310) revealed the presence of a partial gastrectomy without any evidence of jejunal ulcer. There was a

superficial irregularity of the lesser curvature border of the stomach proximal to the site of anastomosis which represented an extension of the original malignant growth.

The postoperative roentgen appearance of an esophagojejunostomy following a total gastrectomy is shown in Figure 311 A and B. Figure 311 A shows the carcinoma of the stomach prior to the total resection. Figure 311 B shows the roentgen appearance of the esophagojejunostomy. At times the segment of the jejunum anastomosed to the esophagus may become unusually dilated. Such abnormal distention associated with an ironing out of the contour due to an obliteration of the valvulae conniventes may produce at times an appearance simulating that of a miniature stomach. It appears almost as if nature, rebelling against the absence of the natural gastric reservoir was attempting by a feeble gesture to overcome this serious deficiency. The appearance following esophagogastrostomy is shown in Figures 105 D and 248 C.

REFERENCES

1. Hellmer H. Étude sur la muqueuse gastro intestinale apres gastro entero anastomose. *Acta radiol* 4 32 1925
2. Barsony T. Beitrage zur Diagnostik des postoperativen jejunalen und Anas tomosenulkus. *Wien klin Wchnschr* 27 1059 1914
3. Zollschan I. Zum roentgenologischen Nachweis des Ulcus pepticum jejuni. *Deutsche med Wchnschr* 44 177, 1918

- 4 Strom, S A contribution to the roentgen diagnosis of *ulcus pepticum jejuni*, *Acta radiol* 2 468, 1923
- 5 Case, J T Gastric and duodenal roentgen-ray findings after operation, *J A M A* 85 1385, 1925
- 6 Camp, J D Jejunal and gastrolejunal ulcer and their associated roentgenologic signs with especial reference to the niche, *J A M A* 91 1436, 1928
- 7 Buckstein, J The niche in the diagnosis of jejunal ulcer, *Am J Roentgenol* 27 59, 1932
- 8 Ledoux-Lebard, R, and García-Calderón, J Le radio-diagnostic de l'invagination jéjuno-gastrique après gastro-entérostomie, *Arch mal app digest* 23 533, 1933
- 9 Included in the paper by A Vespignani *Radiologia delle affezioni dell'intestino tenue*, *Atti d X Cong ital di radiol medica*, Part I (No 3) 1-157, 1932
- 10 Shackman, R. Jejuno-gastric intussusception, *Brit J Surg* 27 475, 1940
- 11 McNamara, W. L Retrograde jejuno-gastric intussusception through a subtotal gastrectomy stoma, *Ann Surg* 120 207, 1944
- 12 Lawson, E H, and Whitener, D L Retrograde jejuno-gastric intussusception Report of case, *Arch Surg* 60 242, 1950
- 13 Grimes, A E Retrograde intragastric intussusception of the jejunum following subtotal gastrectomy, report of case, apparently second such in literature, *Ann Surg* 129 404, 1949
- 14 Aleman, Sten Jejuno-gastric intussusception, *Acta radiol* 29:383, 1948
- 15 Braun, H Demonstration eines Präparates einer 11 Monate nach der Ausführung der Gastro-Enterostomie entstandenen Perforation des Jejunum, *Arch f klin Chir* 59 157, 1899.
- 16 Buckstein, J Roentgenologic evidence of healing of jejunal ulcer, *Am J Roentgenol* 32 487, 1934

THE DUODENUM

The Normal Duodenal Bulb, Including Duodenitis

ANATOMY OF THE DUODENUM

That portion which extends from the pyloric ring to the beginning of the large intestine is the most important part of the alimentary tube. Included here is the extremely roentgenologically significant duodenal bulb, as well as the rest of the duodenal curve and both the jejunum and the ileum. The small intestine is about 6.5 meters long. The lumen becomes gradually smaller as it nears its termination. The duodenum, which is devoid of mesentery, is about 12 fingers in length and derives its name from that fact.

The duodenum consists of the first portion, or duodenal bulb, which lies in close relation to the gallbladder, and of a short second portion, descending in the form of a curve to become continuous with the third or transverse portion, which ordinarily passes across the vertebral column to join the jejunum at the duodenojejunal junction.

Roentgenologically, the first portion of the duodenum, or duodenal bulb, is of the greatest significance, for here occurs the most common organic lesion within the digestive canal—duodenal ulcer. The bulb is about 5 cm. long and is the most mobile part of the entire duodenal curve. It lies in close relation to the quadrate lobe of the liver and the gallbladder.

Of considerable importance in the roentgen anatomy of the second portion of the duodenum are the following two features. It is here that the papilla of Vater, with

its sphincter of Oddi, makes its entrance. Secondly, the curve produced by this second portion frequently hugs the head of the pancreas.

The transverse or third portion of the duodenum lies in front of the inferior vena cava and the abdominal aorta. Anteriorly, it is crossed by the superior mesenteric vessels. This part of the duodenum is thus compressed between the anteriorly and posteriorly placed structures, a point of roentgenologic importance in explaining many of the phenomena of abnormalities of duodenal function.

The fourth portion turns upward and forward to join the jejunum at the duodenojejunal junction.

ROENTGEN DEMONSTRATION OF THE DUODENUM

One of the earliest attempts to demonstrate the duodenum roentgenologically is to be found in the work of Tufner and Aubourg in 1907,¹ who introduced through a gastric fistula in a dog 20 Gm. of subnitrate of bismuth in 100 Gm. of water and then permitted the dog to eat food. They then made fluoroscopic observations of the peristaltic movements in the dog. They also observed the gradual passage of the contents into the duodenum and observed the nature of the segmentation in this region.

The first reference to definite roentgen visualization of the duodenal bulb in the human being is to be found in the work

of Rieder, and particularly in the paper by Schwarz.²

In the roentgen visualization of the duodenal bulb no special technic is necessary ordinarily. Not infrequently the bulb will fill spontaneously the barium leaving the stomach by way of the pyloric ring. More rapid visualization of the bulb may be aided by manual compression of the stomach toward the pylorus. Also, compression of the duodenojejunal area or of the transverse duodenum may prevent the too rapid escape of the opaque meal through the bulb and thereby aid in the clearer visualization of this region.

Roentgenologically, the normal duodenal bulb has a smooth base just beyond the pyloric ring, with an apex distally where it communicates with the second portion of the duodenum. The bulb is cone-shaped with full, rounded, convex margins. Examination may show mucosal strands passing through the pyloric ring up to the base of the bulb. The mucosa of the duodenal bulb itself may be free of folds, but not uncommonly mucosal folds may be noted throughout its length, in many respects similar to those produced by the rugae of the stomach. The same technic applied to the study and visualization of the gastric mucosa applies equally in the recognition of the duodenal relief as well.

Because of its appearance in the roentgenogram and its embryologic, anatomic and physiologic relationship to the stomach, Cole described this portion of the duodenum as the "cap."

The close relationship of the duodenal bulb to the stomach can be explained by their common embryologic origin from the foregut. Anatomically, the pyloric vein acts as a landmark in locating the pyloric ring. In this respect, the screen examination offers a more certain method of demarcation, as the sphincter may be recognized through its physiologic constricting action. Thus, in a borderline case the exact anatomic location of an ulcer as prepyloric or postpyloric can be made with greater assur-

ance through careful roentgen observation than at the operating table by external examination.

Holzknacht³ in 1911, in a review of the progress that had been made up to that time, called attention to the significance of the visualization of the duodenum. He recommended the use of a suspension of bismuth in water instead of the thicker Rieder's meal for the more rapid visualization of the "bulbus duodeni." He believed that the relative width and freedom from folds of this portion acted to prevent the unrestrained hurrying of the ingesta through the remaining portions of the duodenum.

Alvarez⁴ found that when the muscle of the first portion of the duodenum is excised and immersed in Locke's solution, its rhythmicity is less than that of segments of muscle elsewhere in the duodenum. He believed that this weakness of the muscle might explain the fact that, roentgenologically, the duodenal bulb remains filled while the rest of the duodenum is emptying itself rapidly. In human beings no sphincter at the junction of the first and the second portions of the duodenum has ever been described.

DUODENITIS

One of the earliest studies on chronic duodenitis is that of Brioussais⁵ in 1825, who described chronic ulcerations of the duodenum of long standing. These ulcerations may be large and of considerable depth and may be situated in the first portion of the duodenum. They may continue to increase in depth and erode the hepatic artery.

Bailey⁶ made an excellent contribution to the clinical and pathologic findings in duodenitis. He described the inflammatory appearance of the duodenal mucosa, which might become mammillated, and, in cases of long standing, the site of ulcerations which might go on to the stage of perforation. Bleeding from the duodenum might result from the hemorrhagic inflammatory



FIG 312 (A, *Top*) Normal duodenal bulb (B *Bottom*) Note the mucosal folds of the duodenal bulb



FIG 313 Normal duodenal bulb

process or from actual erosion of the hepatic artery. In association with the inflammatory pathology of the duodenum, similar anatomic lesions might also be present in the stomach. He believed that in some cases duodenitis might develop on a nervous basis, "since irritation of the brain is readily transmitted to the duodenum." The association of gastritis with chronic duodenitis was also shown by Baudin.⁷

Gaultier⁸ in his interesting monograph on the duodenum described duodenitis as exudative, suppurative and ulcerative. The last type is associated with the presence of erosions and ulcerations. Like true peptic ulcer, of which he considered these lesions to be an initial stage, they may perforate all the layers of the duodenum with the involvement of neighboring viscera.

Chronic Duodenitis Associated with Ulcer. Puhl⁹ found conditions in the duodenum in association with ulcer resembling those of ulcerative gastritis. He stated that in all cases of gastric and duodenal ulcer he had found an ulcerative gastritis involving chiefly the pyloric glandular area. In some cases of duodenitis, no real ulcer was found in the resected specimens, although the symptoms clinically simulated those of real ulcer.

Judd and Nagel¹⁰ similarly found pathologic evidence of duodenitis in individuals with a history simulating true ulcer. Even roentgenologically, a duodenal deformity was demonstrable, but there was no evidence of a niche. According to Konjetzny, chronic duodenal ulcer forms on the basis of a chronic duodenitis.

Roentgen Manifestations of Duodenitis. Just as efforts have been made to determine the existence of a chronic gastritis roentgenologically, similar attempts have been made, notably by Kirklin,¹¹ toward the roentgen recognition of chronic duodenitis. However, the diagnosis of duodenitis on the basis of the roentgen evidence must be made with considerable circumspection. Kirklin emphasized irritability of the duodenal bulb, irregular reticulation of the

mucosal pattern, absence of an ulcer niche and absence of gastric retention as justifying the diagnosis of chronic duodenitis.

Since the roentgen deformity of duodenitis may closely simulate that of duodenal ulcer, except for the absence of niche formation in the former, this emphasizes the diagnostic value of the niche, since, otherwise, both clinically and roentgenologically, duodenitis and duodenal ulcer may closely simulate each other.

Mere irritability of the duodenal bulb with hypermotility may be of functional origin and not necessarily an indication of organic disease. Moreover, just as we have learned how uncertain the roentgen diagnosis of chronic gastritis can be in the absence of gastroscopic confirmation, so too, apparent abnormalities in the roentgen appearance of the folds of the duodenum must be regarded as only suggestive evidence in the diagnosis of actual anatomic abnormality of the mucosa itself.

It is possible that, with added experience, the only roentgen evidence of duodenitis that will prove to be acceptable will be the persistent presence of rounded translucent areas due to wartlike excrescences of the mucosa, as noted in the stomach. Since the autonomic system as well as allergic response appear to play an important role in the variability of the gastric relief, it is not unfair to assume that these same factors are also capable of producing similar variations in the roentgen appearance of the mucosal folds of the duodenal bulb. Changes in the hydrodynamics of the submucosal fluid may alter the prominence of the folds as observed roentgenologically in the absence of any actual organic disease. It is also possible, on the basis of analogy to the mucosal relief of the stomach, that fine-appearing folds need not necessarily signify an atrophic duodenitis. Moreover, the muscularis mucosae, capable of throwing even an atrophic mucosa into folds in the stomach, should be equally capable of producing an identical phenomenon within the bulb itself.



FIG. 314 Normal duodenal bulb. The base of the bulb partially overlaps the pylorus so that the pyloric ring is obscured.

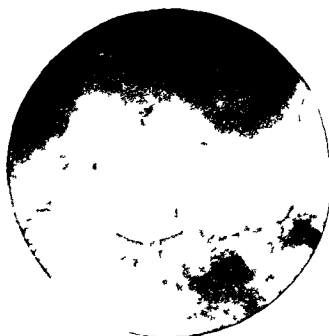


FIG. 315 Base of normal duodenal bulb.



Duodenal Ulcer

EARLY DESCRIPTIONS

One of the earliest descriptions of perforation of what was apparently a duodenal ulcer is contained in an article by Hamberger¹ in 1746

On October 6, 1734, at eight in the morning, a female servant picked up a basket filled with plums and carried it to its destination. Toward evening on returning home, she complained of abdominal pain. On the next morning she had a fever and her abdomen was swollen, and toward evening she died. The possibility of foul play owing to suspicious circumstances made a "judicial autopsy" necessary.

The author's autopsy findings included a round hole which was smaller than a lentil in size, whose margin was turning black and which was situated in the duodenum, six lines ($\frac{1}{2}$ inch) away from the pylorus. This perforation lay in the centre of a scirrhus ring so that the diameter of the entire ring was equal to a finger's breadth or four lines ($\frac{1}{3}$ inch). The middle part of this ring presented a round and almost hemispherical depression about four lines ($\frac{1}{3}$ inch) in diameter covered externally by the "common membrane alone. This membrane was indeed completely pellucid (there being no vestige of either nervous or muscular tunic) and in its centre was located the hole.

Abercrombie in 1824 described a perforating ulcer of the duodenum. A man aged 36 had an attack of violent abdominal pain accompanied by vomiting. The pain gradually became more diffuse, the pulse more feeble, and the patient died. Autopsy revealed the perforation of a duodenal ulcer.

In Figure 2 (Plate 6 installment 38, of his

famous work) Cruveilhier² shows a picture of a simple chronic ulcer of the duodenum which had undergone perforation. He recognized the fact that the first portion of the duodenum was particularly predisposed to such ulceration and that such ulcers might go on to perforation as in the case of ulcers of the stomach.

INCIDENCE AND LOCATION OF DUODENAL ULCER

Most ulcers of the duodenum are near the lesser curvature border. The posterior wall of the bulb is involved much more frequently than was hitherto believed. This is due to the fact that ulcers on the posterior wall may be much more difficult to demonstrate roentgenologically unless careful mucosal technic is employed. Besides, ulcers on the anterior wall are more amenable to clinical recognition, since they are more apt to perforate with the production of peritonitis, than ulcers on the posterior wall. In addition, when the surgeon operates he is more apt to discover an ulcer when it is present on the anterior wall, because he is able to visualize and palpate this region. Unless the duodenal bulb is actually opened up, small ulcers on the posterior wall at the time of operation may escape his notice.

Ulcers on the posterior wall of the duodenum resemble chronic gastric ulcers. As a rule, however, they do not assume as great a depth as the gastric ulcers because they are limited by the posteriorly placed head of the pancreas. They are also less likely to

perforate into the abdominal cavity than the anteriorly placed lesions. When all the coats are destroyed, the floor of the ulcer may then be formed by the pancreas or under surface of the liver.

Another point that detailed inspection of resected specimens has shown is the frequent multiplicity of ulcers of the duodenum. In addition, duodenal ulcers may also have a gastric ulcer as a concomitant finding.

Sturtevant and Shapiro, in their analysis of the Bellevue Hospital autopsy material, showed that of the 44 duodenal ulcers in their series, the percentage of multiple ulcers of the duodenum was 45.45. The duodenal ulcers ranged in size from less than 1 cm to 2.5 cm in diameter, except in one case in which the ulcer encircled the duodenum. One patient had 25 ulcers. Of the total number of duodenal ulcers, 39 were in the first portion, 8 were in the second portion. In all 8 cases, however, there were also ulcers in the first portion as well.

Stewart³ found 23 cases of double ulcer in a series of 74 cases of chronic duodenal ulcer at autopsy. Hauser⁴ found multiple ulcers of the duodenum in about 20 per cent of the cases.

ROENTGEN DIAGNOSIS OF DUODENAL ULCER

The roentgen diagnosis of this disease has been placed upon such a remarkably sound foundation as to offer indisputable objective evidence of its value in the vast majority of cases. It is not unfair to say that it supersedes all other means of arriving at this diagnosis. Extremely important as the clinical history is in the diagnosis of a peptic ulcer, it can give only suggestive aid in the borderline case. It is not at all uncommon for a patient with a definitely atypical history to have a duodenal ulcer and, similarly, for a patient with apparently classical symptoms to show either no evidence of organic disease at all, or when an organic cause is

present, to find it to be an entirely different lesion.

The first roentgen evidence of a character strongly suggestive of the deformity of duodenal ulcer was published by Barclay⁵ in 1910. Skinner⁶ before the American Roentgen Ray Society in September, 1911, had referred to the appearance of an hourglass constriction of spastic nature at the site of duodenal ulcer. Haudek⁷ made his important contributions to the roentgen diagnosis of duodenal ulcer in 1911 and in 1912. He emphasized two main points of diagnosis: (1) the presence of duodenal stenosis resulting from contraction or spasm, (2) the presence of the niche, consisting of a bismuth deposit within the crater of the ulcer. This he considered the most valuable sign because it is the only specific one.

This contribution was followed by the confirmatory studies of Rieder,⁸ Baron and Barsony⁹ and Strauss.¹⁰

Bier¹¹ referred to a case in which a niche with an air bubble was present in the roentgenogram, and at operation an ulcer was found on the anterior superior surface of the duodenum penetrating into the liver.

Kreuzfuchs¹² gave a description of the niche, as a persistent fleck of bismuth, in the beginning of the duodenum, definitely isolated from its surroundings and sharply circumscribed. Although not present in all cases of duodenal ulcer, he considered it nevertheless pathognomonic of the condition when found.

Cole¹³ in 1914 gave a classic description of the essential factors in the diagnosis of duodenal ulcer, depending upon the induration or cicatricial contraction of the duodenal bulb resulting from the presence of the ulcer and producing a constant deformity in the contour of the bulb. In addition, he recognized the roentgenologic significance of the direct visualization of the crater itself and differentiated between the "bullet-hole" and "full-face" types.

The Nature of Duodenal Deformity. Considerable aid in the understanding

of the nature of the duodenal deformity was furnished by the classic monograph of Akerlund¹⁴ and the detailed study of the niche by Berg¹⁵ in 1926

Not uncommonly the deformity of the duodenal bulb in the presence of a duodenal ulcer seems to be guided by no special law. The irregularity appears to be of a bizarre nature, defying detailed analysis of its individual components. On the other hand a duodenal ulcer may exist and yet there may be no detectable evidence of deformity of contour, and only a painstaking search will disclose the presence of a niche, frequently on the posterior wall where it has been obscured by the overlying barium suspension

Between these two types, superficial observation would suggest that the gradations of deformity were well nigh infinite and followed no particular pattern. Closer analysis of the deformity, however, reveals a number of factors which may enter into the sum total of a "classical" duodenal deformity. These factors are niche formation, retraction in the region of the niche, the defect on the curvature opposite, the eccentrically placed, open pyloric lumen, and the diverticular dilatations.

An analysis of these various factors will give one a greater insight into the nature of the duodenal deformity. Not all of these factors will be present in every case. But the greater the number of these factors present in any duodenal deformity the greater the certainty that the deformity was produced by a duodenal ulcer. Moreover, a study of this nature leads to a more intelligent understanding of such secondary manifestations as the occasional development of prestenotic diverticula in association with ulcer.

The following is an analysis of these various features in the deformity of the duodenal bulb which enter into the roentgen diagnosis of ulcer in this region.

The Niche. The niche represents the roentgen counterpart of the ulcer itself.

Originally, the demonstration of the niche was considered to be infrequent, with improvement in technic and more careful search however, the niche was shown with greater frequency. Akerlund, in his fundamental studies, found it in 60 per cent of his cases of duodenal ulcer.

The niche is about the size of a pea although there is considerable variation in this respect. Its most common location is on the lesser curvature border. Even when the ulcer is on the anterior or posterior wall near the lesser curvature, the niche may appear as if projecting directly from the lesser curvature itself. This may be explained according to Forssell, on the basis of a swelling of the mucous membrane in the region of the ulcer, so that the ordinarily convex border of the bulb becomes flattened or actually concave. This brings the lesser curvature border into such close relation to the ulcer that the niche actually appears to project from that border even though the ulcer is some distance away.

As in the case of gastric ulcer, swelling of the mucosal folds plays an important role in the elaboration of the roentgen phenomena directly associated with the niche. The following are the important findings which result from the swelling of the duodenal mucosa at the site of the niche: (1) Owing to a heaping up of the mucosa at the margin of the ulcer, even a superficial lesion may show increased depth in the roentgenogram. (2) The displacement of the barium by the swollen folds of mucosa surrounding the ulcer sometimes produces a translucent halo about the niche. (3) A radiating convergence of the mucosal folds may sometimes be shown as in the case of the niche of gastric ulcer.

In order to demonstrate these various phenomena, a thin layer of barium suspension is essential, with special effort to bring into clear relief the appearance of the duodenal mucosa. In this way not only will the niche which projects from the lesser

ring In a borderline case differential diagnosis may be very difficult or impossible The evidence of a niche may then be of paramount importance for exact localization

As a result of the close approximation of the defect of the greater curvature to the retracted lesser curvature border, an area of stenosis is produced in this region This may in the early stages be due entirely to functional changes, such as contraction of the muscular fibers and swelling of the mucosal folds In later forms this stenosis is exaggerated and made permanent because of cicatrization There are two important results which follow because of the obstruction produced thereby (1) ultimate enlargement of the stomach itself, (2) the production of prestenotic pseudodiverticula in the region between the base of the bulb and the narrowed zone, as described by Hart. This is produced in the following manner Gastric peristalsis carries its contents through the pyloric ring into the duodenum There the material meets with an obstruction to its onward flow at the stenosed area The proximal portion of the duodenal bulb therefore begins to dilate Eventually, if this process is continued over a sufficiently long period and the stenosis remains unyielding or even becomes progressive, the dilatation may assume the appearance of an actual diverticulum of the pulsion variety This is most frequently noted at the junction of the base and greater curvature border of the duodenal bulb Such diverticular dilations may retain barium for a long time after the rest of the barium has left the stomach and the duodenum The diverticulum of the lesser curvature may in some cases be confused with the actual niche However, its characteristic location, its occasional ability to alter its contour and the absence of the mucosal phenomena seen in association with ulcer will all help in differentiating this finding from niche formation

The classical picture of the duodenal de-

formity of an ulcer may therefore include (1) the niche, (2) the defect of the greater curvature opposite, (3) the retraction of the lesser curvature at the site of the niche, (4) the eccentric position of the pyloric ring, and (5) pseudodiverticula in that portion of the bulb proximal to the area of stenosis Not all these features can, of course, be shown in every case of ulcer A knowledge of the pathologic physiology underlying the formation of these various types of deformity, however, will aid considerably in an understanding of those factors which justify a diagnosis of ulcer, particularly when the niche is absent At times, the deformity of the bulb may appear to follow no particular law The irregularity may be extreme yet it may still not be possible to demonstrate in any clear-cut fashion the operation of the various factors which have been considered This may be due to the predominance of scar formation of an irregular character, to the presence of multiple ulcers with atypical deformities, or to repeated insults by a succession of lesions over a long period of time, each one of which may have left some telltale evidence of its original presence

While the niche of an ulcer is ordinarily associated with a deformity of duodenal contour, such deformity is not present in every case The bulb, in exceptional cases, may have a perfectly normal outline and, when completely filled with barium, may exhibit no evidence of disease It is under such circumstances that the demonstration of the niche becomes a matter of prime importance Detailed mucosal studies, with compression for the demonstration of the niche, then assume their greatest significance in order to bring a posteriorly placed niche into clear relief On the other hand, when the deformity of bulbar contour becomes extremely marked, the bulb shows an extreme narrowing of the lumen, with a diminution in size, the "phthisis bulbi" of Freud

A number of other signs of a suggestive

character have been described as secondary aids in the diagnosis of ulcer of the first portion of the duodenum. On the whole, however, these are of only minimal value as compared with those deformities already considered. Thus Chaoul has described an elongation of the pyloric canal due, in part, to contraction of the distal portion of the pyloric antrum and of the proximal portion of the duodenal bulb. As evidence of a functional nature, it may show no permanence in the various films or on repeated examination. Certainly in the absence of changes of a more direct character, the diagnosis of ulcer would hardly be based on such a finding alone.

Still another roentgen finding, considered by some of the continental writers as suggestive of duodenal ulcer, is the so called "Dauerbulbus" or permanent filling of the bulb at a time when the stomach is empty. It is possible that some of these cases were confused by some of the older writers with persistent retention within a pseudo diverticulum of the prestenotic variety. Such saccular formations, particularly at the junction of the base and the greater curvature of the bulb, may be quite large, at times appearing to be about the size of a duodenal bulb. It requires careful examination and an attempt to force the barium through the area of stenosis into the distal portion of the duodenal bulb in order to arrive at a definite differentiation. It is quite possible, therefore, that delayed emptying of such a sacculus may have been confused with an actual delay in the emptying of a permanently filled bulb. When the entire bulb itself remains filled this evidence cannot be accepted as an indication of ulcer in the absence of other findings. It may be an entirely functional phenomenon and re examination may fail to show its recurrence.

Localized tenderness at the site of the duodenal bulb is by itself a sign of little value. Combined with other evidence of deformity of the duodenum it may in some

cases add further certainty to the original diagnosis.

Another suggestive evidence of disease of the duodenal bulb is diminution of mobility, as determined by manual palpation under fluoroscopic examination. In the case of an ulcer that is adherent to adjacent structures the duodenal bulb may be fixed in position. However, only a slight degree of mobility of the duodenal bulb may be present even under normal conditions. Moreover, an examination of this character may be difficult when the individual is stout or when the position of the bulb is high.

Another secondary evidence of some suggestive value is the extreme rapidity in the passage of the barium through the duodenal bulb, so that complete filling is difficult and at times practically impossible. Manual compression of the pyloric antrum in the direction of the duodenal bulb with the right hand of the examiner, while pressure is exerted against the second portion of the duodenum with the left hand, may aid in complete distention of the bulb and the recognition of the nature of its contour. In some cases, the very rapid transit of the barium through the bulb may arouse suspicion as to the irritable nature of this region as the result of inflammatory disease. So many other factors, such as the nature of the peristaltic activity of the stomach, the gaping of the pyloric ring and possibly the character of the gastric secretion may be involved, that definite conclusions based on the occurrence of this phenomenon cannot be made in the absence of confirmatory findings. Moreover, what is apparently an exaggerated emptying of the duodenal bulb at the onset of the examination may eventually become more normal in the later stages. Possibly some of these cases may represent intermediate stages in the life history of the ulcer in the actual process of development from its earliest incipency in the spastic process to its final transformation into the full blown lesion.

Reflex spasm in the stomach in the pres-

ence of a duodenal ulcer may show itself in several ways (1) One may, in rare cases, note an incisura of the greater curvature of the stomach (2) Much more frequently, there is spasm of the pyloric antrum (3) Spasm at the pyloric ring may take place, and actual hypertrophy of the pyloric muscle may occur Such a finding plays a definite role in the eventual production of secondary gastric enlargement, hyperperistalsis and all the later eventualities that arise from a progressive and uncompensated type of pyloric obstruction Finally, a suggestive type of spastic manifestation in the stomach is to be found in the appearance of the mucosal folds¹⁶ These frequently appear unusually prominent and tortuous throughout the entire stomach from cardia to pylorus The appearance may be suggestive of an actual chronic gastritis and yet be fundamentally an expression of reflex irritation In the absence of definite evidence of organic gastric disease, such an appearance of the mucosal relief may be highly suggestive of the presence of a duodenal ulcer In some cases, evidence of an actual gastritis may be present in association with duodenal ulcer, as previously described

Within the duodenal bulb itself there may be increased prominence of the mucosal folds in the presence of a duodenal ulcer as an expression of functional irritability or of an actual duodenitis (Puhl, Konjetzny) In the absence of niche formation or any other of the more characteristic deformities of bulbar contour, such unusual prominence of the duodenal relief picture may in some cases justify the diagnosis of duodenitis without the presence of an actual ulcer Just as care must be taken in the evaluation of the roentgen appearance of the gastric folds as evidence of chronic gastritis, so considerable circumspection must also be employed in the interpretation of an organic inflammatory duodenitis In the case of the duodenal bulb, however, the salutary check of the gastroscopist is unfortunately lacking

Other Causes of Deformity of the Duodenal Bulb. While the most common cause of a defect of the duodenal bulb is the presence of an ulcer, such defects may, in rare instances, be of extrinsic origin At times, a smooth indentation may be noted near the apex of the duodenal bulb under normal conditions, due to the pressure of the common duct on its way to the papilla of Vater Also, a smooth concavity of the greater curvature border of the bulb may be due to the pressure of a normal gallbladder Such evidence of pressure is exaggerated when the gallbladder is enlarged because of disease or is the site of malignant degeneration In addition to pressure defects, duodenal deformity may arise as a reflex phenomenon due to gallbladder pathology

Other deformities of the duodenal bulb are produced by the presence of intrinsic masses Among these are papillomas and polyps As previously described, polyps originating on the gastric side may appear as defects within the duodenal bulb because of the fact that they have been intussuscepted through the pyloric ring as a result of gastric peristalsis Deformities of the duodenal bulb due to primary cancer in this region have been described, but these must at best be exceedingly rare and for all practical purposes they can be excluded from differential diagnosis A carcinoma of the pancreas may break through into the duodenal bulb and produce a deformity in this region

It is because there occasionally may be other causes for the production of a deformity of the duodenal bulb that it becomes so important to demonstrate the actual niche of the ulcer itself as the ultimate diagnostic criterion

Pyloric Obstruction Resulting from Duodenal Ulcer The gastric enlargement in postpyloric obstruction causes the stomach to extend to the right Also, owing to the fixation of the duodenal bulb, the pyloric antrum extends beyond this region Hyperperistalsis may be one of the early phenomena and may compensate for pyloric



FIG 318 "Classical" duodenal ulcer deformity. Note (1) The niche on the lesser curvature border (2) The shortening of the lesser curvature to either side of the niche (3) The smooth incisura of the greater curvature opposite (4) The eccentric pyloric ring.



FIG 319 Niche of a duodenal ulcer, with shortening of the lesser curvature and incisura of the greater curvature

obstruction, so that no serious impairment of motility is noted. Such peristalsis may, as in pyloric obstruction from other causes, show itself characteristically in two ways

(1) by an increase in the number and depth of the waves and (2) by the fact that these waves start high up at the cardiac end of the stomach. Finally, one may also note occasional evidence of antiperistalsis, of alternating periods of hyperperistalsis and complete cessation of gastric activity, with the eventual development of a large, bag-like atonic type of stomach characteristically half moon in appearance with re



FIG 320 (A, Left) Niche within the duodenal bulb not visualized (B, Right) Same patient. The niche was visible at this time with thin layer method

tention at 24 and 48 hours and even longer

In late stages of obstruction it may be impossible to demonstrate any portion of the duodenal bulb. The stomach, considerably enlarged, will, however, exhibit no evidence of intrinsic disease. This will aid in placing the obstructive lesion at or beyond the pyloric ring. In some cases, however, a correct anatomic localization of the cause of the obstruction may be impossible. All that may then be justified on the basis of the objective roentgen evidence is the diagnosis of pyloric obstruction of organic origin.

It must be remembered, however, that here also spasm and edema may be contributory factors in addition to actual scar formation in the production of obstruction at the pyloric ring. Only a therapeutic test with a course of medical management will lead to a determination as to the predominance of these various factors. Even in cases with a surprising degree of obstruction and 24- and even 48-hour retention, examination after medical treatment may show a return to a normal size and normal emptying of the stomach. Obviously, spasm and edema at the pyloric ring are the important elements in such cases of obstruction.

Illustrative Cases. A "classical" deformity of the duodenal bulb produced by an ulcer is illustrated by the following case.

C. H., male, aged 49. The patient gave a history of recurring episodes of epigastric pain of 5 months' duration. The pain was worse about 2 hours after eating and was eased by food and antacids. It became progressively more intense, at times radiating into the right lower quadrant and the back. He lost 10 pounds in the preceding month.

Physical examination was essentially negative, except for tenderness in the epigastrium and the right lower quadrant.

Roentgenographic examination (Fig. 318) revealed an ulcer deformity of the duodenal bulb exhibiting the following features: (1) a niche on the lesser curvature border of the bulb, (2) shortening of the lesser curvature to either side of the niche, (3) a smooth indrawing of the greater curvature opposite

the niche, (4) eccentricity of the pyloric ring, favoring the shortened lesser curvature border.

Operation revealed an ulcer on the anterior surface of the first portion of the duodenum just distal to the pylorus. A subtotal gastrectomy was done.

Macroscopic examination. "The gastric mucosa is essentially normal. Just distal to the pylorus is a shallow ulcer surrounded by a fairly thick rim of fibrous tissue."

Pathologic diagnosis. Chronic ulcer of the duodenum.

The significant features entering into the deformity produced by a duodenal ulcer are illustrated further by the following case.

C. T., male, aged 37. The patient gave a 3-year history initiated by an acute perforation of an ulcer without preceding symptoms. After operative closure of the perforation, he remained well for about 2½ years when he again had a perforated ulcer which was sutured. One month later epigastric pain recurred. Physical examination was essentially negative except for the scars of the previous surgical procedures.

Operation revealed an ulcer on the anterior wall of the first portion of the duodenum. A partial gastrectomy was done.

Roentgen examination (Fig. 319) revealed a cone-shaped niche on the anterior wall near the lesser curvature border of the duodenal bulb. There was shortening of the lesser curvature border in this region and an incisura of the greater curvature opposite. It is noteworthy that in spite of two former acute perforations, the patient again had a recurrence of duodenal ulcer with a niche.

The most reliable criterion in the diagnosis of duodenal ulcer is, of course, as has been stressed in the text, the demonstration of the niche, the roentgen counterpart of the ulcer itself. Such visualization of the niche may be of prime diagnostic importance when the bulb itself exhibits no clear evidence of deformity. This is illustrated in Figures 320 A and B (P. C., aged 24). Figure 320 A shows the duodenal bulb completely filled with barium. There is certainly no evidence of any niche. With a thin layer of barium, however, a large, centrally located niche is clearly visible. The niche



FIG 321 (*Left*) Niche of a duodenal ulcer. Note the halo surrounding the niche, apparently the result of the protrusion of the swollen wall surrounding the ulcer.
 FIG 322 (*Right*) Large niche of a duodenal ulcer.

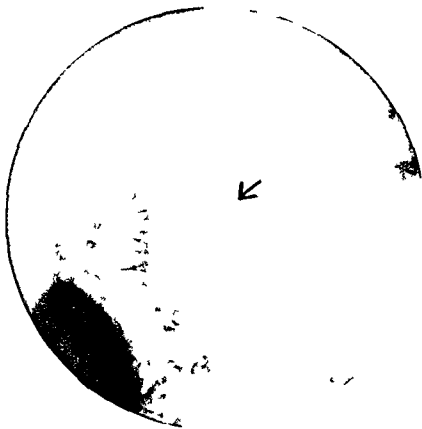


FIG 323 Note the niche partly surrounded by a halo within the confines of the duodenal bulb. The contour of the bulb itself is essentially intact.



FIG 324 Niche of duodenal ulcer When the bulb was completely distended with barium, the niche was not visible, and the contour of the bulb was normal



FIG 325 Niche of duodenal ulcer When the bulb was filled with barium, the niche was not visible, although the contour was irregular By means of graded compression, the niche was brought clearly within the field of vision and fixed in the roentgenogram by means of spot-film technic



FIG 320 (A, *Left*) Duodenal ulcer. No evidence of the niche at this time (B, *Right*) With graded compression of the duodenal bulb and spot film technic the niche is brought into clear relief

itself is surrounded by a halo, apparently due to the swollen mucosa surrounding the ulcer (Fig 320 B)

The halo about a niche, due to edema of the surrounding wall, is particularly well shown in Figure 321. Note also the deformity of both the lesser and the greater curvature borders

Although the niche of a duodenal ulcer is usually smaller than that of a gastric ulcer it may nevertheless assume considerable size as may be noted in Figure 322. The contour of the bulb itself is deformed

The value of the thin layer method in the demonstration of a niche which would otherwise escape detection is illustrated by Figures 323, 324 and 325. Spot film technic with graded compression is of considerable value in thinning out the barium sufficiently to bring the niche clearly into the field of vision

The value of graded compression and spot film technic in the demonstration of a niche is also illustrated by the case of G. N., female, aged 38, who gave a typical ulcer history. With the usual contour study of the

stomach and the duodenum (Fig 326 A) there is no evidence of any niche. There is a slight deformity of the lesser curvature border of the duodenal bulb. When the examination of the duodenal bulb was made by means of graded compression and spot film technic the niche of the ulcer crater was readily demonstrable (Fig 326 B)

Not only is exploration of the confines of the duodenal bulb important in order to demonstrate the niche which may otherwise be obscured, examination through every angle of obliquity may also aid in the successful visualization of a niche which might escape detection

J. G., male, aged 40. The patient gave a classic duodenal ulcer history over a period of 7 years. During the preceding 3 months the pain had become continuous. He had lost 15 pounds and the stools were positive for blood. Surgical intervention was therefore decided upon

At operation a firm indurated duodenal ulcer was found about 2 cm distal to the pylorus which extended posteriorly so as to become attached to the pancreas. There was a considerable amount of moderately edematous

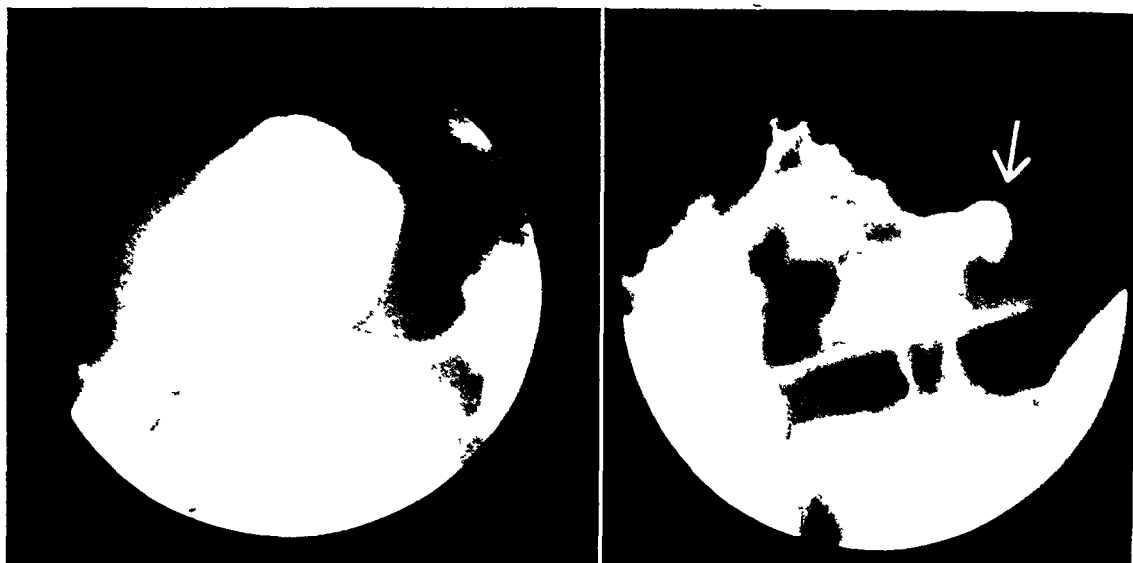


FIG 327 (A, *Left*) Duodenal ulcer the niche is not visible (B, *Right*) Same patient Examination in right oblique position shows the niche at the border of the lesser curvature

scar tissue surrounding the ulcer. There were a few fine adhesions to the fundus of the gallbladder. A partial gastrectomy was done.

Roentgen examination (Fig 327 A) when the duodenal bulb was filled with barium and viewed at an angle of 90 degrees showed no definite evidence of abnormality. When the patient was examined in the first, or right oblique, position the niche was clearly visible (Fig. 327 B).

In addition to the diagnostic significance of the niche as the major criterion in the diagnosis of duodenal ulcer, in many cases it is possible to determine the exact anatomic location of the ulcer from the position of the niche. If the ulcer is on the posterior wall of the duodenal bulb, then examination in the right oblique position will show the niche as projecting from the greater curvature border of the bulb. If the ulcer is on the anterior wall of the duodenal bulb, then this same maneuver (examination in the right oblique position) will project the niche from the lesser curvature border of the bulb. This is illustrated by the following case:

E. M., male, aged 52. The patient gave a 20-year history of recurring episodes of indigestion relieved by the ingestion of milk or antacids. Shortly before hospitalization his

symptoms became worse, and he had a tarry stool. Physical examination revealed a pale white male. Abdominal examination was essentially negative, except for slightly increased resistance in the right upper quadrant and mild epigastric tenderness.

Radiographic examination (Fig 328 A and B) in the right oblique position showed a deformity of the duodenal bulb. In addition, a niche was noted originating from the greater curvature side of the duodenal bulb. Because of the fact that the niche projected in this manner, it was stated that the ulcer crater responsible for the niche was on the posterior wall.

At operation: "An ulcer was seen involving the first inch of the duodenum just distal to the pyloric vein. The ulcer mass penetrated posteriorly into the substance of the pancreas."

Macroscopic examination revealed "a 3-mm ulcer crater on the posterior wall and on the duodenal side of the pylorus. The base of this ulcer is hard and sclerotic, but no definite blood vessel can be detected."

Pathologic diagnosis: Chronic ulcer of the duodenum.

In this case, therefore, the fact that the niche projected from the greater curvature border of the duodenal bulb when examined in the right oblique position made it possible not only to make a diagnosis of duodenal ulcer but also to localize it accurately to the posterior wall. In the event that direct inspec-



FIG 328 A and B Ulcer on the posterior wall of the duodenal bulb Examination in the right oblique position Note that in addition to the deformity of the duodenal bulb there is a persistent niche projecting from the greater curvature border This made it possible to state that the ulcer crater was on the posterior wall of the duodenum



FIG 329 A and B Ulcer on the anterior wall of the duodenal bulb Examination in the right oblique position Note that the niche is projected from the lesser curvature of the duodenal bulb (B Bottom) In A (Top) the niche was not visualized because of lack of sufficient distention of the bulb with barium



FIG 330 Unusually large niche due to an ulcer on the posterior wall of the duodenal bulb.

tion and palpation of the bulb at the time of operation does not disclose the duodenal ulcer, then the roentgen recognition of its location will put the surgeon on guard, so that if necessary he may incise the anterior wall and directly inspect the posterior area

The very reverse situation existed in the following case:

E V., male, aged 54 This is the same patient a description of whose case may be found on page 169 Figure 112 as an example of an ulcer of the esophagus Examination also revealed an ulcer of the first portion of the duodenum (Fig. 329 A and B taken in the right oblique position). Note that in addition to the deformity of the duodenum, the niche extends from the lesser curvature border of the duodenal bulb Based on this finding the ulcer crater was localized to the anterior wall The location of the ulcer was fully confirmed at operation when a subtotal gastrectomy was done

This case is also of interest in that in Figure 329 A the niche is not seen clearly, although it is definitely demonstrable in Figure 329 B. This is due to the fact that in Figure 329 A, the bulb was not sufficiently distended with barium at the moment when the roentgenographic exposure was made Therefore although a niche may actually be obscured when the bulb is com-



FIG 331 Note the niche corresponding to the ulcer at about the junction of the first and the second portions of the duodenum

pletely filled as has been illustrated previously, it is also possible for a niche to escape recognition unless the bulb is actually distended with barium Contour studies as well as investigation of the mucosal "relief" must supplement each other if we are to obtain a maximum degree of efficiency in the demonstration of the actual niche

The anatomic location of an ulcer of the duodenal bulb may also be predicted, if the niche is unusually large This is illustrated by the following case.

A W, female, aged 53 The patient gave an 8-year history of recurrent episodes of epigastric pain usually about 1½ hours after meals She vomited blood on several occasions During the preceding 2 months the epigastric pain was most marked at night

Physical examination of the abdomen was essentially negative

Roentgenographic examination revealed an unusually large niche, occupying the region of the duodenal bulb (Fig 330) Because of the large size of the niche it was assumed that the ulcer was on the posterior wall Ulcers on the anterior wall practically never reach



FIG 332 Ulcer niche at the junction of the first and the second portions of the duodenum. In addition to the niche note the constriction of the lumen at that site.



FIG 333 Translucent area within deformed bulb, due to ulcer with a thrombosed blood vessel at its base.

such size. Any exaggeration particularly in their depth will lead to perforation. On the posterior wall of the duodenal bulb, however, the spread of the lesion is prevented from perforation by adhesions which fix it to neighboring structures.

At operation a large ulcer was found on the posterior wall of the duodenal bulb. A subtotal gastrectomy was done.

The localization of an ulcer at the distal end of the duodenal bulb is also made possible by the demonstration of the niche.

A J. male aged 44. The patient gave a 6 year history which was characterized by one episode of massive gastrointestinal hemorrhage at the onset and a similar recurrence 3 years later. Several months before his last admission to the hospital he complained of epigastric pain radiating to the lumbar spine. The morning that he was admitted he had two tarry stools. He had gained 50 pounds in the preceding 2½ years.

Physical examination of the abdomen was essentially negative.

Roentgenographic examination (Fig 331) revealed evidence of an ulcer niche at about the junction of the apex of the duodenal bulb and the second portion of the duodenum. Note the marked constriction of the lumen at the

site of the niche. The presence of the ulcer was confirmed by surgical intervention at which time a subtotal gastric resection was done.

Another example of the niche of a duodenal ulcer occurring at the junction of the bulb with the second portion of the duodenum is illustrated by the following case.

E. N. aged 34. Six years previously the patient experienced severe substernal pain. This was relieved by eating and by the ingestion of bicarbonate of soda. He had periodic recurrences until finally an episode of pain and vomiting of blood brought him to the hospital. He responded to medical management only to have a recurrence a few months later of pain in the epigastrium radiating to the left chest and back. He vomited daily. No blood was present in the vomitus at this time. There was no history of tarry stools. The pain finally became so severe that he again sought hospitalization.

Physical examination was essentially negative.

Operation revealed the following: There was a thickened cicatrized ulcer of the duodenum. The ulcer area was examined through a rent in the transverse mesocolon and it was shown to involve the posterior surface of the duodenum as well as the anterior.

Roentgenographic examination (Fig 332) revealed a niche at the distal portion of the duodenal bulb near its junction with the sec-

ond portion of the duodenum There was considerable narrowing of the duodenum at this site The niche extended beyond the inner border of this narrowed region The stomach itself was normal There was a 6-hour gastric residue of about two fifths of the barium

This case again demonstrates two salient roentgen findings in ulcer at the junction of the first and the second portions of the duodenum, namely, the niche and the constricted area of the duodenum at the site of the niche

As a rule, the crater of an ulcer is fairly homogeneously filled with barium A central zone of translucency may be noted, however, in the event that the crater is occupied by a mass such as a blood clot or a rigid blood vessel This is illustrated by the following case:

P S, male, aged 60 The patient gave a 2-year ulcer history associated with secondary anemia due to blood loss At the time of his last admission to the hospital, he had had severe bleeding Particularly because of the persistent blood loss and the severe anemia, surgical intervention was decided upon A subtotal gastrectomy was done and reported on as follows "There was an ulcer approximately 1.5 cm in diameter situated on the antero-lateral aspect of the first portion of the duodenum A thrombosed vessel was seen in the base of the ulcer"

Roentgen examination (Fig 333) showed a marked deformity of both the lesser and

the greater curvature borders of the duodenal bulb Within the confines of this deformity was a small, rounded translucent area, which was interpreted as representing the crater surrounded by a rim of barium The failure to visualize the central area was considered to be due either to a blood clot or a rigid blood vessel As noted at operation, the center of the ulcer was occupied by a thrombosed blood vessel This condition explained the central translucency in the niche

The mechanism involved in the production of a central translucent zone within the niche of a duodenal ulcer is illustrated by the findings at autopsy in a patient who died of massive hemorrhage At autopsy, there was a large ulcer of the duodenal bulb and within it the rigid blood vessel from which the uncontrolled bleeding had occurred A barium suspension was injected into the blood vessel leading into the ulcer and the surface was coated with a thin layer of barium (Fig 334) Note the centrally located density within the ulcer due to the barium-filled blood vessel The degree of central translucency within a niche would then vary with the relative size of the blood vessel as compared with the total size of the crater

A duodenal niche associated with radiating mucosal folds of the duodenum is illustrated by Figure 335

An important deformity developing in the



FIG 334 Illustrating the relation of the blood vessel to an ulcer by injection of barium suspension into the blood vessel



FIG 335 Niche of duodenal ulcer, showing radiating mucosal folds



FIG 336 Prestenotic diverticulum secondary to duodenal ulcer



FIG 337 Prestenotic diverticulum (indicated by black arrow) secondary to duodenal ulcer. The niche of the duodenal ulcer is indicated by the white arrow

progress of a duodenal ulcer is that of the prestenotic diverticulum. This is illustrated by the following case:

F. D., male, aged 30. Fourteen years previously this patient had developed vague abdominal pain. At that time he had had a hemorrhage, with vomiting of bright red blood and the passing of blood in the stools. He made a good clinical recovery and was well until 5 years later when he had another hemorrhage. A third hemorrhage occurred 1½ years before his admission to the hospital. In addition he had had recurring episodes of abdominal pain relieved by food. His last recurrence had been 8 weeks prior to his admission. It was similar to the pain he had had with previous attacks. There was no hemorrhage at this time. Physical examination was essentially negative except for slight tenderness in the epigastrium.

The report at operation: "There was an ulcer just beyond the pyloric ring associated with a false diverticulum. There was a marked pancreatitis and marked edema between the duodenum and the head of the pancreas. A subtotal gastrectomy was done."

Roentgen examination (Fig 336) revealed a marked deformity of the duodenal bulb with a centrally located niche. In addition there was a very large pouch formed by the proximal portion of the greater curvature and the base of the bulb. Both the ulcer and this

pouchlike prestenotic diverticulum were found at operation as described. Note that the incisura of the greater curvature at its mid portion has caused a marked degree of obstruction in this region. As a result of this obstruction there has been a gradual dilatation of the duodenum proximal to this region by the chyme which after entering from the stomach was unable to pass readily through the zone of obstruction. In this way the pseudodiverticulum or prestenotic diverticulum seen in the roentgenogram gradually developed.



FIG 338 Niche of a duodenal ulcer associated with two prestenotic diverticula. The niche is centrally located. The diverticula are to either side.

Another excellent example of a prestenotic diverticulum is to be noted in Figure 337. The large cone-shaped niche is centrally located. A mucosal fold radiates from it both proximally and distally. At the junction of the greater curvature border and the base of the bulb is a comparatively large diverticulum, which is prestenotic in origin, developing as a secondary manifestation proximal to the obstruction produced by the ulcer.

Figure 338 shows the niche of a duodenal ulcer in association with two prestenotic diverticula at the junction of the base with both the medial and lateral borders.

S. S., male, aged 45. The patient gave an 18-year history of recurring attacks of epigastric pain relieved by food, usually coming on in the spring of the year. He vomited coffee-ground material at times, and the stools were black.

Physical examination of the abdomen was essentially negative. Operation revealed a large ulcer in the first portion of the duodenum which had eroded through to the head of the pancreas and was adherent to this region. A partial gastrectomy was done.

Roentgen examination (Fig 338) showed



FIG 339 Duodenal ulcer. Narrowing of the duodenal bulb at the site of the ulcer. Note the gaping pyloric ring.

a marked deformity of both the lesser and the greater curvature borders of the duodenal bulb. Centrally located and just distal to the pyloric ring, was a large niche representing the actual ulcer. There was a saccular collection of barium to either side of the bulb, at the junction of the base with the greater and the lesser curvature borders. These two areas represented prestenotic diverticula secondary to the more distally placed ulcer.

The same factor that is responsible for the development of prestenotic diverticula, namely narrowing of the duodenal bulb at the site of the ulcer, also may be the cause of a gaping pyloric ring.

Enlargement of the stomach and abnormal gastric retention may then be secondary to the constriction within the duodenum itself. The fact that the pyloric ring is wide open shows that the real offender must be sought in the more distally placed constricted area of the bulb. This is illustrated by the following case.

A. S., male, aged 58. The patient gave a 5-year history of recurrent attacks of hunger pain. He responded well at first to medical treatment. Finally the pain became intractable and he vomited brownish-colored fluid. A subtotal gastrectomy was done for duodenal ulcer.

Roentgen examination (Fig 339) showed

a deformity of the duodenal bulb. There was a large niche near the lesser curvature border and a defect of the greater curvature opposite thereby constricting this portion of the bulb. The pyloric ring was unusually wide and eccentrically placed. At 6 hours there was a

gastric residue of about one third of the barium. Delay in the emptying of the stomach was obviously not due to any abnormality of the pyloric opening but could be more rationally explained on the basis of the constriction within the bulb itself.

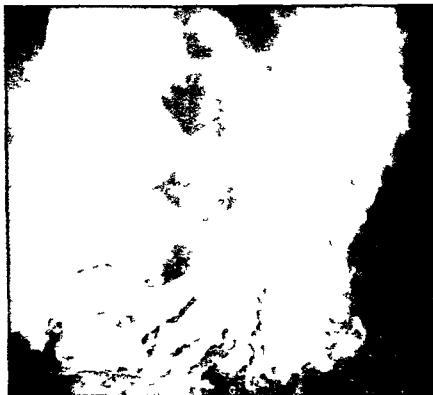


FIG. 340 Unusually prominent gastric mucosal folds in a patient with duodenal ulcer.



FIG. 341 (A *Left*) Apparently normal duodenal bulb. (B *Right*) Same patient. Two niches are demonstrable in the partly filled bulb.



FIG 342 Gastric enlargement secondary to duodenal ulcer

Figure 340 shows the presence of abnormally prominent gastric mucosal folds in a case of duodenal ulcer. Such an appearance may be of functional origin, due to edema and swelling of the mucosa secondary to the presence of duodenal ulcer. However, a marked degree of prominence in the mucosal folds may also be the expression of an actual chronic gastritis.

The following is an example of multiple ulcers of the duodenal bulb.

J. D., male, aged 24. This patient complained of an acute episode of epigastric pain, which "doubled him up" accompanied by nausea and the vomiting of a large quantity of coffee-grounds material. Physical examination revealed abdominal rigidity and rebound tenderness in the right upper quadrant.

At operation, a large, indurated ulcer was found on the anterior surface of the first portion of the duodenum which was plastered against the under side of the right lobe of the liver. It was believed that this ulcer might have perforated against the liver but there was no evidence that the ulcer had perforated into the peritoneal cavity. When the lesser sac was opened, an area of thickening about the size of the end of the thumb was felt in the duodenum, which could not be separated from the head of the pancreas.

Roentgen examination showed that when the duodenal bulb was completely distended with barium, there was no evidence of any niche formation (Fig 341 A). The contour of the duodenal bulb was essentially normal. When the bulb was only partially filled (Fig 341 B), two large niches were demonstrable within it, one apparently corresponding to the perforated duodenal ulcer of the anterior wall and the other represented an ulcer on the posterior wall attached to the head of the pancreas.

Occasionally, multiple niche formation, instead of being visualized "en face," as in Figure 341 B may be readily demonstrable by their projection from the lesser and greater curvatures of the bulb.

An important complication of duodenal ulcer is obstruction with secondary gastric enlargement and abnormal gastric retention. This is illustrated by the next case.

K. S., female, aged 45. This patient had been complaining of pain in the right lower quadrant (worse at night) during the preceding 8 years. Five years previously she had had an appendectomy. She had occasional vomiting, which on one occasion was brownish-red. Physical examination was essentially negative.

The report at operation was: "There was an old duodenal ulcer with practically a complete stenosis at the pylorus. A gastro-enterostomy was done."

Roentgen examination (Fig 342) showed the stomach to be increased in size, extending well over to the right. There was exaggerated peristaltic activity of both the greater and the lesser curvatures. The contour of the stomach was intact. Only a small amount of barium was present in the duodenal bulb, which appeared to be markedly constricted. At 6 hours there was a gastric residue of one-half of the barium.

The diagnosis was duodenal ulcer, with obstruction and marked secondary gastric enlargement. The gastric enlargement, the exaggerated peristalsis and the abnormal gastric retention are all secondary to the postpyloric (duodenal) ulcer.

Pyloric obstruction secondary to ulcer may be the result of actual unyielding scar tissue, or it may be due to edema, inflammation and spasm, or the cause may be the result of the play of all these factors combined. That other factors besides scar-tissue production do operate in producing

pyloric obstruction is illustrated by the next case

A S, female, aged 24. The original examination of the stomach in this case disclosed it to be of markedly increased size (Fig 343 A). The duodenal bulb could not be clearly visualized because of the difficulty of forcing barium into this region. There was a gastric residue 24 hours after the administration of the barium.

Roentgen examination 5 months later showed the stomach to be of normal size (Fig 343 B). Not only was there no retention of barium in the stomach at 24 hours, but there was none at 6 hours. In this examination barium escaped from the pylorus readily. The duodenal bulb was clearly visualized and exhibited a deformity characteristic of ulcer in this region. Since all evidence of pyloric obstruction had disappeared, factors of an evanescent nature, such as edema, inflammation and spasm must have played an important role in the production of the findings in the original examination.

DUODENAL ULCER BEYOND THE BULB

Duodenal ulcer beyond the bulb itself is quite rare. When it does occur, it involves

the second portion of the duodenum. Roentgenologically, involvement of the second portion of the duodenum by an ulcer is recognizable (1) by the presence of a niche, similar in appearance to that of a niche within the duodenal bulb itself, and (2) by the presence of marked narrowing of the second portion of the duodenum in the region of the niche, with considerable alteration of the mucosal relief in the involved area. In addition, tenderness localized in the niche gives further support to the diagnosis. Because of the unusual location of the lesion, there is a possibility of confusion with a duodenal diverticulum, the latter being a far more common occurrence anywhere in the duodenal curve beyond the bulb itself.

Differential diagnosis will depend first on the presence of a narrow neck of sharply defined contour in the presence of a diverticulum. Normal strands of mucosa may be seen passing from the duodenum into the neck of the diverticulum. In addition, the most common location of a diverticulum of the second portion of the duodenum is in

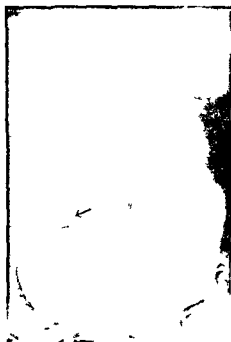
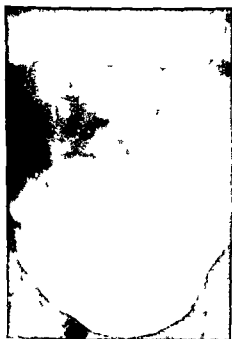


FIG 343 (A Left) Marked gastric enlargement secondary to duodenal ulcer (B Right) Same patient 5 months later, showing the normal size of the stomach.

the periampullary region. A more detailed description of the appearance and behavior of duodenal diverticula will be found in a chapter devoted to that subject.

The niche of an ulcer of the second portion of the duodenum is most apt to occur just beyond the duodenal bulb. It does not possess a neck, which is so characteristic a finding in diverticula, but projects directly from duodenal contour. Of considerable importance is the associated marked narrowing of the adjacent duodenum, with evidence of destruction of the normal mucosal markings, due to the inflammatory process. A history suggestive of a duodenal ulcer and perhaps of blood in the stool will lend additional support to the diagnosis of a peptic ulcer of the second portion of the duodenum.

The combination of a niche with narrowing of the lumen as evidence of ulcer of the second portion of the duodenum has been corroborated in the contributions of Via,¹⁷ Wolke,¹⁸ Bignami¹⁹ and Garin and Bernay.²⁰

The only example that I can find in the literature of a peptic ulcer of the duodenum originating just below the papilla of Vater

with roentgen confirmation was reported by Kaiser.²¹ Roentgenographically, a large sacular area was noted in the region of the papilla which closely simulated the appearance of a diverticulum. However, the duodenum between the bulb and this region was markedly narrowed, strong evidence of inflammatory pathology. The roentgen findings were corroborated both by surgery and autopsy.

Illustrative Case The following is an example of ulcer of the second portion of the duodenum.

M. M., male, aged 52. This patient had suffered from indigestion at various intervals since childhood. He complained of burning pain, often radiating to the middle of the back and relieved by eating and the taking of soda. In addition, he had apparently similar pain in the precordium, in the right and left shoulders and in the left arm and jaw, this occurred frequently after exertion and was associated with palpitation of the heart. There were periods of remission in the epigastric distress lasting for months. Physical examination of the abdomen was negative except for slight tenderness in the epigastric region.

At operation the following report was made: 'On entering the peritoneal cavity there are



FIG. 344 Ulcer involving the second portion of the duodenum.

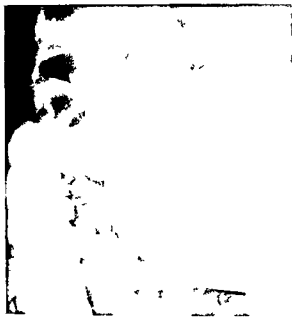


FIG 345 Ulcer at the beginning of the second portion of the duodenum, characterized by the niche and the constriction of the lumen at that site

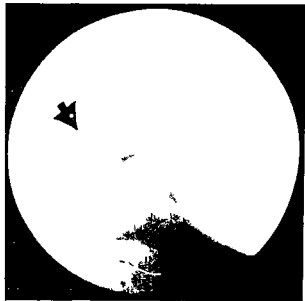


FIG 346 Niche of an ulcer of the second portion of the duodenum

adhesions of the omentum, duodenum and stomach which are easily separated and an ulcer is easily demonstrated. The ulcer extends from the first portion of the duodenum down to the second portion of the duodenum and is adherent to the pancreas. In view of the patient's general condition and the extent of the ulcer, it was deemed wise to do only a posterior gastro enterostomy."

Roentgen examination (Fig 344) revealed an area of marked narrowing just beyond the duodenal bulb. Distal to this was a large well rounded niche occurring in the proximal region of the second portion of the duodenum. The roentgen diagnosis was ulcer involving the proximal area of the second portion of the duodenum.

A surgically corroborated case of ulcer of the second portion of the duodenum is that of J. L. male, aged 20.

The patient gave a 5 year history of a burning sensation in the epigastrium more pronounced at night about 3 to 4 hours after the evening meal. It was not relieved by food intake. During the preceding 3 months he vomited every day. He made no favorable clinical response to an ulcer regimen. Three days before hospitalization he passed a tarry stool. He lost 10 pounds in the preceding

6 months. Physical examination was essentially negative.

Roentgenographic examination (Fig 345) showed a niche at the junction of the first and the second portions of the duodenum. The lumen at this site was constricted. Operation confirmed the roentgenographic findings. The ulcer was on the anterior superior surface at the beginning of the second portion of the duodenum.

Another example of the niche of an ulcer of the second portion of the duodenum is illustrated by Figure 346. Note also the constriction of the lumen of the second portion of the duodenum in the region of the niche.

This patient (W. C. male, aged 50) gave a classical history of ulcer with intermittent episodes of epigastric distress, food aversion and night pain over a period of many years. When examined during periods of remission of the symptoms there was complete disappearance of the niche, only to recur with a return of clinical symptoms. It is also of interest in this case that over a period of years the niche did not always occupy the same location. This variation in the location of the niche suggested that the ulcer was not always the same one from which he had made a clinical recovery previously.

A niche of the second portion of the

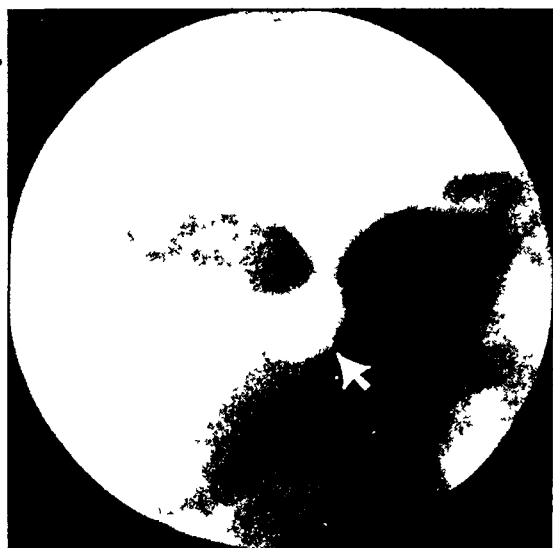


FIG 347 Ulcer deformity of the second portion of the duodenum. Note the constriction of the lumen of the duodenum just distal to the bulb and the niche extending from the inner border.

duodenum just distal to the bulb is also illustrated in the case of S C, male, aged 45.

The patient gave a 15-year history of recurring attacks of epigastric pain which responded to ulcer therapy. On one occasion he had a tarry stool. Physical examination except for marked obesity was negative.

Roentgenographic examination (Fig 347) shows a classical ulcer deformity of the second portion of the duodenum. Just distal to the duodenal bulb is an area of marked narrowing with a niche extending from the inner border.

The characteristic roentgen features in the diagnosis of ulcer of the second portion of the duodenum are (1) evidence of the niche beyond the duodenal bulb itself, and (2) usually, as in the above cases, an area of marked narrowing of the second portion of the duodenum in the region of the ulcer.

COMBINED GASTRIC AND DUODENAL ULCER

Kienbock²² reported one of the earliest examples of combined gastric and duodenal ulcer. An hourglass constriction of the stomach noted on roentgen examination was not found at operation.

Roentgen evidence of combined gastric and duodenal ulcer has been found in our experience at Bellevue Hospital to occur approximately once in 1,500 examinations of the stomach and duodenum for upper abdominal distress. As previously noted in the survey by Sturtevant and Shapiro of the autopsy material at Bellevue Hospital, in 86 cases of open gastric ulcer there were 4 in which an open duodenal ulcer was also found.

The following is an example of combined gastric and duodenal ulcer.

A B, male, aged 69. About 4 weeks before admission to the hospital the patient had a sudden attack of sharp, cramplike pain in the epigastrium which radiated up the right side of the chest to the right shoulder and lasted several minutes. From then on, the pain recurred at almost any hour of the day or night. He had had a similar episode the preceding year, which had lasted about 3 weeks. He had lost 38 pounds.

Physical examination of the abdomen was essentially negative except for tenderness in both upper quadrants.

Operation revealed a gastric ulcer on the posterior wall of the stomach about 4 inches below the esophagus which had penetrated into the tail of the pancreas. In addition, there was an ulcer on the anterior superior surface of the duodenum. A total gastrectomy was done with esophagojejunal anastomosis. Pathologic examination revealed no evidence of malignancy.

Roentgen examination (Fig 348) in the first, or right oblique, position showed a niche high up on the posterior wall near the lesser curvature, pars cardiae. The lesser curvature to either side of the niche was of comparatively smooth outline. In addition, there was a deformity of the first portion of the duodenum, with a niche at the lesser curvature border. The preoperative diagnosis was combined gastric and duodenal ulcer. It was my opinion that the gastric ulcer was benign, not only because there was no evidence of associated malignant infiltration in the region of the ulcer but also because there was an associated duodenal ulcer. A carcinomatous ulcer of the stomach is quite rare in the presence of a duodenal ulcer.

The following case of combined ulcer of the stomach and the duodenum was confirmed by autopsy.

J K, male aged 53 The patient's illness was of $1\frac{1}{2}$ years duration His main complaints were dyspnea, orthopnea, occasional ankle edema and epigastric pain He had lost 20 pounds in the preceding 6 months There was no history of melena or hematemesis The epigastric pain radiated to his back and occasionally to his left shoulder and arm There was no definite relation to meals Sometimes the pain was relieved by rest or antacids The clinical impression on physical examination was that the patient was in congestive heart failure There were rales at the bases of both lungs The ap \times of the heart was in the fifth left interspace almost in the anterior axillary line The liver was enlarged 3 fingers breadth below the right costal margin There was a 3+ pitting edema of the ankles and severe anemia The blood pressure varied between 220/130 and 190/108 Gastric analysis showed marked hyperacidity

Roentgenographic examination (Fig 349) revealed evidence of a niche of the lesser cur



FIG 349 Combined gastric and duodenal ulcer confirmed at autopsy, at which time a second duodenal ulcer was also found



FIG 348 Combined gastric and duodenal ulcer

vature of the stomach which was considered to be due to a benign gastric ulcer. In addition the niche of a duodenal ulcer was also present. The patient's course was gradually downhill, his dyspnea became more marked and he died.

At autopsy the essential cause of death was 'arterial and arteriolar nephrosclerosis, with hypertrophy and dilatation of the heart and chronic passive congestion of the lungs, the liver and the spleen.'

Examination of the stomach revealed the following: "Upon opening the stomach it contains considerable gas and approximately 800 cc of dark-brown and black fluid. The mucosa is injected and the rugae are prominent. There is a large punched-out ulceration on the lesser curvature approximately 6 cm proximal to the pylorus. This appears to extend through the musculature but not the serosa of the organ. It measures 1.7 cm in diameter. Upon opening the duodenum there are two large ulcerations immediately distal to the pyloric ring: one is of a typically punched-out appearance, measuring 1.5 cm in diameter, and the other is somewhat triangular in shape with approximately the same diameter."

Microscopic examination showed the lesions to be benign.

Note that although the gastric ulcer and one duodenal ulcer were found on roentgen examination, the second duodenal ulcer was discovered only at autopsy.

A further example of combined gastric and duodenal ulcer is illustrated in Figure 350.

PEPTIC ULCER IN INFANCY AND CHILDHOOD

Peptic ulcer in infancy and childhood is a comparatively rare disease although, particularly since the advent of more universal radiologic study of the alimentary tract in childhood, reports of cases are finding their way into the literature in increasing numbers. Cruveilhier²³ described gastric ulcers in three infants and included the drawings.

Helmholz²⁴ in 1909 stated that duodenal ulcer in infancy was more common than had been supposed and he included descriptions of 8 infants in which the diagnosis was confirmed at autopsy. He considered a thrombosis of the vessels of the duodenum as the direct pathologic cause of the development of the lesion. Shortly thereafter²⁵ he included 7 additional cases of duodenal ulcer, a rare experience. In view of this large number of cases, it seems unusual that he was able to find only 16 recorded cases of duodenal ulcer in children in a review of the literature up to that time.

A survey of the world literature on peptic ulcers in children up to 1919 may be found in the thorough treatise of the subject written by Thiele.²⁶

Stutevant and Shapiro²⁷ in their analysis of the autopsy material at Bellevue Hos-

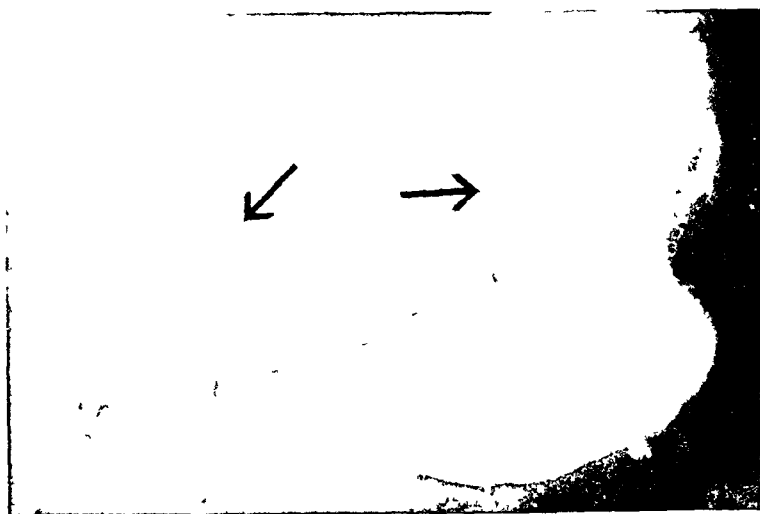


FIG. 350 Combined gastric and duodenal ulcer

pital between 1904 and 1922 found 5 cases of gastric ulcer in children under 9 years of age and 1 case of duodenal ulcer in that group

Excellent reproductions of the pathologic appearance of peptic ulcer in infants are to be found in the reports by Nixon and Fraser,⁸ Rogers,⁹ Guthrie¹⁰ and Franklin.³¹

Foshee³ reproduced the roentgenogram showing the niche of a gastric ulcer in a boy of 13 confirmed by surgical intervention

An unusually large number of ulcers in children was described by Bigelow.³² He reported 10 cases with excellent roentgen demonstrations of the lesion. It is rather surprising that he found these 10 cases out of a total of 186 cases of duodenal ulcer in a period of 20 months, representing an incidence of 5 per cent. The ages of the children cited in his study varied from 9 to 15 years.

Fisher's case (Fisher³⁴) was of a male infant 8 months old. Autopsy revealed 7 independent ulcers of the duodenum. Multiple ulcers of the duodenum were also found in an infant 4 months old, in a child 3½ months old, and in another 6 weeks old. In two of the cases an ulcer had perforated, causing a localized peritonitis. In one case ulcers occurred both proximal and distal to the papilla of Vater. In the other 3 infants the ulcers were proximal to the papilla. In none of the cases was there an associated gastric ulcer or any other disease. The infants were all boys.

It is important to keep in mind the possibility of a peptic ulcer in infancy and childhood because in its symptomatology and complications the lesion behaves as in the adult. It may be the cause of massive hemorrhage and prompt surgical intervention may be successful (Plummer and Stabins³). Acute perforation may be recognized by air under the diaphragm on roentgenographic examination as in one of my cases of duodenal ulcer in a child.

A remarkable complication of gastric ulcer in a newborn was described by Pein.³⁶

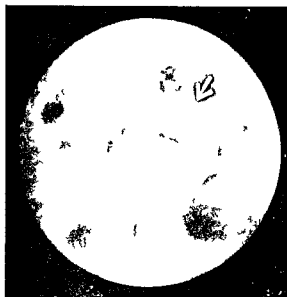


FIG. 351 Duodenal ulcer in a child of 7

Roentgenographic examination following the administration of barium by mouth in this 5 week old male baby showed a gastrocolic fistula. Operation and later autopsy showed a chronic ulcer of the stomach which had perforated into the colon. I have had one personal experience with a case of spontaneous gastrocolic fistula in an adult at Bellevue Hospital resulting from a primary gastric ulcer which had perforated into the colon. (There had been no prior surgical intervention.)

With a recognition of the fact that peptic ulcer may be more common in infants and children than had hitherto been supposed, roentgen examination has been resorted to more frequently. As a result, there has been an increasing number of reports in the literature of gastric and duodenal ulcer in the very young.

Illustrative Case. The following is a case of duodenal ulcer in childhood.

E. T., male, aged 7. The child's presenting symptoms at that time were vomiting at night after his evening meal and some difficulty in swallowing. A few months later he developed abdominal cramps, occurring from 1 to 2 hours after meals and relieved by food. The pain finally became continuous day and night without any relief. Roentgen examination at that time (Fig. 351) showed a deformity char-

acteristic of ulcer of the first portion of the duodenum. The patient responded to medical management but was readmitted to the hospital from time to time because of recurrent attacks of pain. The pain at times woke him from sleep at night. On one occasion, he vomited a considerable amount of blood. Also on one occasion, at the age of 11, he developed a sudden attack of very severe upper-abdominal pain 18 hours before his admission to the hospital. By the time he reached the hospital the pain had somewhat subsided.

Physical examination showed right upper quadrant spasm and deep tenderness.

Roentgen examination at that time showed a collection of air between the liver and the right diaphragm. However, the acuteness of the attack appeared to be subsiding and no surgery was attempted. The child made an uneventful recovery.

This, then, is the story of a child who at the age of 7 had classic roentgenographic evidence of a duodenal ulcer as well as a typical history. He had had some abdominal discomfort and vomiting 2 years before and it is possible that roentgen examination at that time might have disclosed the lesion. The child later had a hemorrhage and after that an episode of acute perforation, with air under the diaphragm, from which he recovered without surgical intervention. About 1 year and 9 months later the child again had a recurrence of epigastric pain which subsided under medical management.

THE NICHE AS A CRITERION IN THE HEALING OF DUODENAL ULCER

Not only is the niche the most reliable criterion we possess in the diagnosis of ulcer, but it may also serve as the best gauge of the effect of medical management. Because of the increased depth of the niche produced by the swollen mucosal folds, there may be, as in the case of gastric ulcer, a rapid diminution in size in the roentgenogram after the institution of treatment. This is due to the prompt diminution in the degree of edema and swelling in the wall itself.

One of the first evidences of the healing of an ulcer may be the gradual disappearance of the light zone surrounding a niche, due to a diminution in the elevation of the

surrounding mucosa. Also, following hemorrhage, the elevated wall of mucosa may be considerably diminished, thereby decreasing the depth of the niche to such a degree as to make it recognizable with difficulty in the roentgenogram.

The disappearance of the niche within a short time, therefore, does not necessarily mean that the ulcer itself has undergone complete healing. There are other interesting features in connection with roentgen studies of the niche which may be noted in the course of treatment. Following the original disappearance of the niche, there may later be a recurrence paralleling the return of manifestations of clinical activity. Thus a life cycle in the history of the niche may be demonstrated. Food, mucus or a blood clot may occlude the ulcer so that a niche cannot be seen, owing to the inability of barium to penetrate the lesion. Owing to marked swelling of the mucosa surrounding the ulcer, the area of communication between the duodenum and the ulcer itself may be so completely closed off as to prevent its radiologic visualization, even though the lesion is both clinically and pathologically in a state of activity. Even when the niche itself is no longer visible, the radiation of the mucosal folds may remain as an evidence of its original presence.

When a careful technic is employed, with examination by the thin-layer method, the disappearance of a niche of the duodenum coincidental with clinical amelioration of symptoms may be accepted as a reliable, although far from infallible criterion for the determination of the actual healing of the ulcer itself.³⁷ The value of such evidence of disappearance of the niche is considerably enhanced if the niche remains persistently absent on repeated examinations over a comparatively long period of time. The reason why the duodenal niche is the only acceptable criterion in determining a successful therapeutic response is that the duodenal deformity itself, other than the niche, may be unyielding in spite of retrogressive changes in the actual ulcer itself.

Such a deformity of contour when the pre dominating factor is the laying down of scar tissue may be permanent in nature and may remain with the patient indefinitely, like the deformity produced by scar tissue elsewhere. The deformity may therefore be silent evidence of an ulcer that has long since healed and must not be considered as indicating that a clinically active ulcer is present. When the original evidence of the presence of a duodenal ulcer consists only in the roentgen demonstration of irregularity of contour, we have no reliable feature which we may call into service to enable us to determine whether the ulcer has actually healed. This is one of the reasons, among others, why a thorough and painstaking search of the duodenum should be made for the recognition of a niche, even though the irregularity of the bulb may be entirely sufficient from a purely diagnostic point of view.

As already stated, there may be a puckering of the mucosa, with coronal radiation of the folds, to mark the site of an ulcer of which the niche has disappeared in the healing process. Another finding is that of a rigid lesser curvature border having the appearance of a straight line at the site of the original niche. There may be no irregularity in this contour in spite of the fact that this loss of roundness of contour is apparently due to a cicatrizing process. In many respects it is similar to the short, rigid straight, abruptly terminated peristalsis free area of the lesser curvature of the stomach, which may, in rare instances, mark the site of the niche of a gastric ulcer which has disappeared in the healing process.

Another point of some importance is the matter of whether a reappearance of symptoms is necessarily paralleled by the recurrence of a duodenal niche as originally present. The answer is that at times these two phases may be independent of each other. That is to say, a patient who showed a characteristic niche of a duodenal ulcer when he first came for roentgen study may, long after, fail to show the reappearance of

the niche even though there has been a return of symptoms. A possible explanation is that the symptomatic reactivation may be due to a new but very superficial ulcer which does not show as a niche in the roentgenogram, or to the presence of a gastro duodenitis without any progress of the lesion beyond the stage of superficial erosion.

That duodenal ulcers do heal has been demonstrated by Sturtevant and Shapiro in their analysis of the Bellevue Hospital autopsy material. They concluded that about 20 per cent of duodenal ulcers showed pathologic evidence of having been healed. Such pathologic proof of duodenal ulcer healing is also to be found in the original paper by Hart.³⁸ He had established beyond question the fact that such healing, with scar formation, actually does take place. He also described the origin of the prestenotic diverticulum of the duodenum which developed in association with ulcer. In some cases the mucosa may be so completely restored in the healing process of duodenal ulcer that only the slightest deviation from the normal remains. The muscularis mucosae, however, is destroyed as a rule and such destruction may be recognized on microscopic examination. Such evidence is therefore of importance in the demonstration of an ulcer that had previously existed. Only in the case of a simple erosion may healing leave no evidence of its original presence.

Illustrative Cases. The following case shows the disappearance of the niche of a duodenal ulcer after medical management.

J. O., male, aged 23. This patient had periodic recurrences of epigastric distress for a period of 3 years. The longest interval of freedom from distress prior to this examination had been 6 months. The distress was epigastric, did not radiate and occurred a few hours after meals. It was relieved by food. He had never vomited. His bowel function was irregular but he had never noticed blood. He had not lost any weight. He gave no history of night pain. Physical examination revealed tenderness in the right upper quadrant.

Roentgenographic study showed the stomach to be normal. No pathologic retention was

present Within the duodenal bulb a niche was demonstrable This was persistently present during prolonged fluoroscopy and on repetition of the examination It is also shown in the accompanying roentgenogram (Fig 352 A) The patient responded readily to medical treatment, with complete disappearance of all symptoms and a moderate gain in weight

Two years after the first roentgen study, during which time the patient felt well, he returned for re-examination At this time the most careful study, fluoroscopically and with numerous films, failed to reveal the niche which had been present (Fig 352 B)

A number of years later, he had an acute perforation of his duodenal ulcer which required surgical intervention

When a niche is demonstrable, its disappearance may be noted as a check upon the efficacy of medical treatment Roentgenologic study thus serves as a valuable means of indicating objective progressive changes in duodenal ulcer

That the niche of a duodenal ulcer may be used as a criterion to indicate healing by the demonstration of its diminution or complete disappearance is further illustrated by the next case

A H, female, aged 26 This patient had been complaining of epigastric distress for 1½ years. She had had periodic remissions of about 1 month's duration, only to have the symptoms recur in a more aggravated form. The pain occurred from 2 to 3 hours after meals, was localized to the epigastrium, did not radiate and was relieved by food She was occasionally awakened about 2 or 3 o'clock in the morning with pain lasting for a few minutes Once she was awake all night with pain She vomited twice, but never vomited any blood At one time she noticed blood in the stool She had lost 20 pounds in weight Physical examination was negative except for epigastric tenderness

Roentgenographic examination revealed the presence of a persistent niche within the duodenal bulb The stomach itself was normal

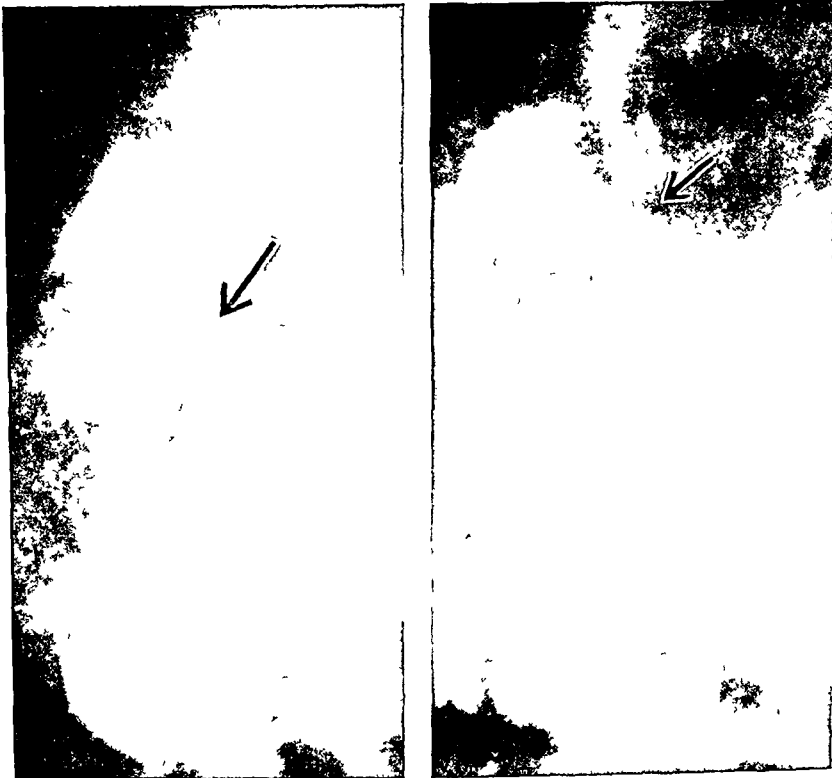


FIG 352 (A, *Left*) Niche of a duodenal ulcer (B, *Right*) Same patient There is no evidence of the original niche 2 years later

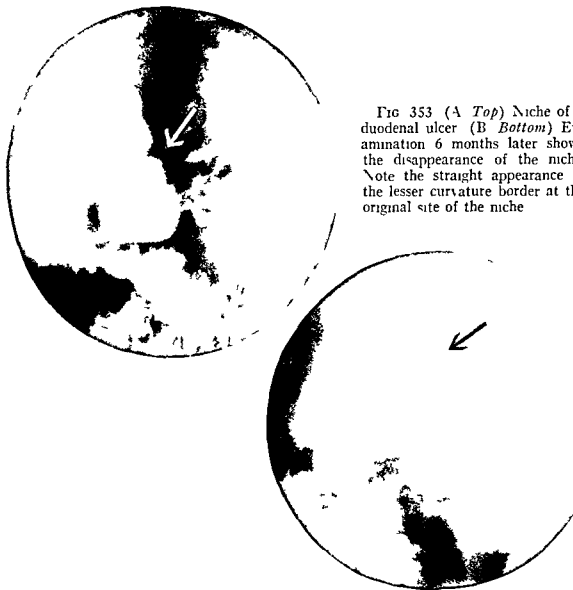


FIG 353 (A *Top*) Niche of a duodenal ulcer (B *Bottom*) Examination 6 months later shows the disappearance of the niche. Note the straight appearance of the lesser curvature border at the original site of the niche.

No gastric retention of the motor meal was present at 6 hours. A diagnosis of duodenal ulcer was made. The appearance of the niche is clearly seen in the accompanying roentgenogram (Fig 353 A). Note the light area surrounding the niche, apparently due to elevation of the encircling mucosa.

The patient was placed on medical treatment. Her symptoms disappeared. She regained the 20 pounds she had lost. Six months after the first roentgenogram she was again examined. In spite of the most careful fluoroscopic examination, which was repeated several times with particular emphasis on manual pressure over the bulb, as well as with numerous films, the niche originally noted was no longer at all demonstrable (Fig 353 B).

The lesser curvature border of the duodenal

bulb, however, is straight, the usual curved appearance no longer being present. This finding was persistent on repeated examinations. It may therefore be accepted as evidence of stiffening of the lesser curvature border of the duodenal bulb as the result of organic cicatricial infiltration secondary to ulcer. There is another point of considerable importance here. This patient was followed over a period of several years. During this time, she had periodic recurrences of symptoms identical with those she had had at the time of the original examination when a niche was found in the duodenal bulb. In spite of apparent clinical recurrence, however, examination during these periods failed to disclose a recurrence of the original niche. Therefore, although disappearance of the niche of an ulcer is excellent objective evi-

dence of clinical amelioration, this is not necessarily true in all cases. The recurrence of symptoms in the absence of a definite niche may possibly mean that an acute erosion or a duodenitis may have developed which was not roentgenologically demonstrable.

The disappearance of the niche of a duodenal ulcer coincidental with the disappearance of clinical symptoms is well illustrated by the following case.

P C, female, aged 27. This patient gave a 3-month history of hunger pain, with food relief and night pain. Physical examination revealed exquisite tenderness to the right of the epigastrium.

Roentgen examination showed the presence of a large niche within the duodenal bulb (Fig 354 A). One year after the institution of medical management, roentgen examination failed to reveal any evidence of the original niche (Fig 354 B). The patient made a good clinical recovery. Several years later she had a recurrence of clinical activity. Examination at that time, however, again failed to show any evidence of recurrence of the niche, although radiating mucosal folds were demonstrable, evidently secondary to cicatrization.

Like peptic ulcer elsewhere in the stomach or duodenum, an ulcer of the second portion of the duodenum may undergo regression under medical management. Such gradual diminution in size will obviously be a reliable criterion in differential diagnosis between an ulcer and a diverticulum in this region.

Roentgen evidence of the gradual disappearance of a niche of the second portion of the duodenum is illustrated by the following case.

A R, male, aged 45. The patient gave a 26-year history of recurring attacks of epigastric pain, occasionally radiating to the back and relieved by food and antacid medication. The pain frequently kept him awake during the night. There was no vomiting or bleeding or weight loss. Physical examination was essentially negative except for tenderness in the epigastrium.

Roentgen examination (Fig 355 A) showed narrowing of the second portion of the duodenum just distal to the bulb, with a destruction of the normal mucosal markings. There was a niche at the inner border of this involved area. Figure 355 B about 2 weeks after

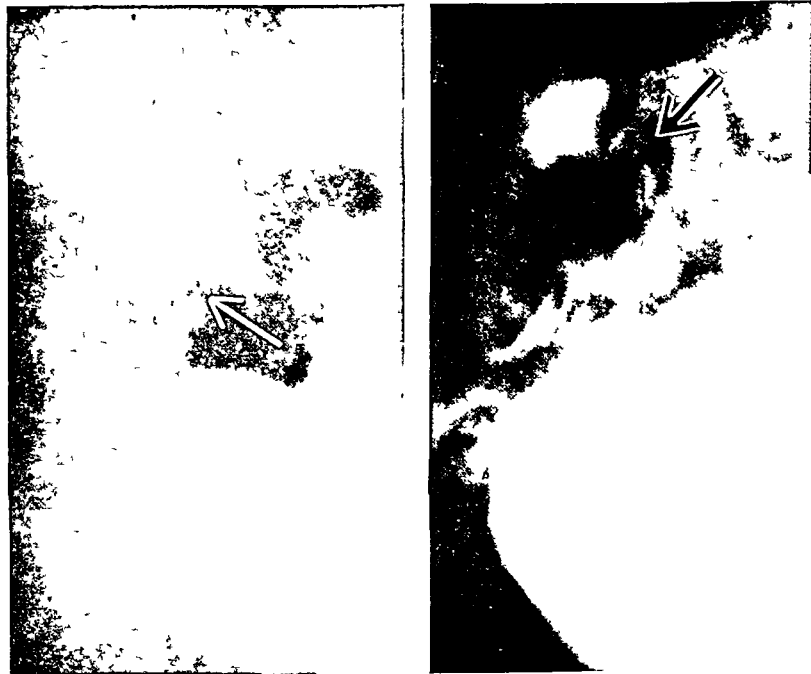


FIG 354 (A, *Left*) Niche of a duodenal ulcer (B, *Right*) Same patient. Note the disappearance of the niche one year later.



FIG 355 (A, Left) Ulcer in second portion of the duodenum (B Center) Same patient Appearance 2 weeks after medical treatment Note the diminution in the size of the niche (C, Right) Appearance 6 months after medical treatment, showing that the original niche is no longer evident However, there is considerable narrowing and irregularity of the second portion of the duodenum in this region

medical treatment showed a definite reduction in the size of the niche Figure 355 C about 6 months later showed a disappearance of the niche There was, however, persistent narrowing of the involved second portion of the duodenum with destruction of the normal mucosal markings The clinical improvement paralleled the roentgen evidence, and at the time of the disappearance of the niche the patient was quite well The patient, however later continued to have recurrences of clinical activity and about $1\frac{1}{2}$ years later roentgen examination showed a niche in the second portion of the duodenum

Because of recurrent episodes of pain hemorrhage and obstruction, he was finally operated and the lesion resected

FREEDOM FROM MALIGNANT DEGENERATION OF DUODENAL ULCER

The remarkable immunity of duodenal ulcer to the development of malignancy is shown by the fact that in a series of over 26 000 autopsies at Bellevue Hospital there is no record of a single case of carcinoma arising in a duodenal ulcer This, of course is one of the reasons why considerable doubt is entertained regarding the peptic ulcer origin of malignancy in general However a variation in the environmental factors within the duodenum as compared with the stomach may perhaps explain the fact that malignancy is sometimes believed to be

engrafted on an originally benign ulcer when this is present in the stomach

It is of great clinical significance to know that, once the ulcer has been definitely limited to the postpyloric region, malignancy as a complicating factor may be ruled out completely

This freedom of duodenal ulcer from malignant change is also shown in the contributions of other writers^{3 40 41}

ROENTGENOGRAPHIC EXAMINATION OF THE UPPER DIGESTIVE TRACT DURING HEMORRHAGE

An important problem sometimes arises as to the advisability of making roentgenograms of the upper digestive tract in the presence of hemorrhage Often such an investigation may be extremely useful It is assumed of course that the patient is not in such a state of profound shock that any additional movement such as putting him on the stretcher would prove to be hazardous Abdominal manipulation such as is ordinarily done during fluoroscopy is also contraindicated With these precautions in mind the administration of a barium suspension may be considered as quite safe It is customary in many cases to feed a patient promptly during the course of a massive hemorrhage as part of medical management It is quite likely that a simple

REFERENCES

- 1 Hamberger, G E De ruptura intestini duodeni, Jenae, Dec 17, 1746 in A Haller Disputationes ad morborum historiam et curationem facientes, Lausanne, v 3, pp 507-509, 1757
- 2 Cruveilhier, J Anatomie pathologique du corps humain, Paris, 1835-1842.
- 3 Stewart, M J: The morbid anatomy of gastric and duodenal ulcer, Internat Clin 334 1, 1923
- 4 Hauser, G Das chronische Magengeschwür, Handbuch der speziellen pathologischen Anatomie und Histologie, IV, Part 1, F Hanke and O Lubarsche, Berlin, 1926
- 5 Barclay, A E The normal and pathological stomach as seen by the x-rays, Brit M J 2 537, 1910
- 6 Skinner, E H Duodenal diagnosis, J A M A 57.1308, 1911
- 7 Haudek. H Die Diagnose des Ulcus duodeni im Röntgenbilde und seine Unterscheidung von Ulcus pylorum, Verhandl deutsch Gesellsch Chir 40 192, 1911
- 8 Rieder, H Das Röntgen-Verfahren im Dienste der Pathologie und Therapie des Magendarmkanales, Verhandl deutsch Gesellsch inn Med 29 17, 1912
- 9 Baron, A, and Barsony, T Spastischer Sanduhrmagen bei duodenaler Affektionen, Wien klin Wchnschr 25 1185, 1912
- 10 Strauss, H Das Duodenalulkus und seine Feststellbarkeit durch Röntgenstrahlen, Fortschr Geb Röntgenstrahlen 19 461, 1913
- 11 Bier, A Über das Ulcus duodeni, Deutsche med Wchnschr 38 788, 836, 1912
- 12 Kreuzfuchs, S Zur Technik des Röntgenuntersuchung des Duodenalgeschwures, Berlin klin Wchnschr 49 1568, 1912.
- 13 Cole, L G The diagnosis of postpyloric (duodenal) ulcer by means of serial radiography, Lancet 1 1239, 1914
- 14 Akerlund, A Röntgenologische Studien über den Bulbus duodeni, mit besonderer Berücksichtigung der Diagnostik des Ulcus duodeni, Acta radiol, supp 1, 1921
- 15 Berg, H H Die direkten Röntgen-symptome des Ulcus duodeni und ihre klinische Bedeutung, Ergebn Med Strahlenforsch 2 249, 1926
- 16 Buckstein, J The gastric "relief" in duodenal ulcer an accessory aid in diagnosis, Am J Digest Dis & Nutrit 5 81, 1938
- 17 Via, E Contributo alla conoscenza radiologica della ulcera seconda porzione del duodeno, Arch ital mal app diger 4 44, 1935
- 18 Wolke, K Über Ulzera in der Pars descendens duodeni, Acta radiol 17 371, 1936
- 19 Bignami, G Sull'ulcera extrabulbare del duodeno, Quaderni radiol. 3 207, 1932
- 20 Garin, C, and Bernay, P Trois cas de duodénum en M avec ulcère duodénal loin du bulbe, Arch mal app digest 27 193, 1937
- 21 Kaiser, R Beitrag zur Differentialdiagnose echter und unechter Duodenaldivertikel, Röntgenpraxis 9 538, 1937
- 22 Kienbock, R Zur radiologischen Diagnose der Magen- und Darm-Erkrankungen, Wien med. Wchnschr 62 1050, 1912
- 23 Cruveilhier, J Anatomie pathologique du corps humain, 1829-1835
- 24 Helmholz, H F J Über Duodenalgeschwüre bei der Padatrophie, Deutsche med Wchnschr 1 534, 1909
- 25 ——— The relation of duodenal ulcers to atrophic conditions of infants, Arch Pediat 26 661, 1909
- 26 Thiele, P Ueber Geschwürsbildungen des Gastro-Duodenaltractus im Kindesalter, Ergebn inn Med u Kinderh 16 302, 1919
- 27 Sturtevant, M, and Shapiro, L L Gastric and duodenal ulcer, Arch Int Med 38 41, 1926
- 28 Nixon, J A, and Fraser, A D Peptic ulcer in the newborn, Arch Dis Childhood 3 157, 1928
- 29 Rogers, J S Y Duodenal ulcers in two infants of the same family, Arch Dis Childhood 3 163, 1928
- 30 Guthrie, K J Peptic ulcer in infancy and childhood with a review of the literature, Arch Dis Childhood 17 82, 1942
- 31 Franklin, A White Two cases of

- duodenal ulceration in children, Arch Dis Childhood 3 95, 1942
- 32 Foshee, J C Chronic gastric ulcer in children—report of a case, J A M A 99 1336, 1932
 - 33 Bignami, G L'ulcera duodenale nell'età giovanile, Radiol med 26 394, 1939
 - 34 Fisher, J H Duodenal ulcer in infants Am J Dis Child 79 50 1950
 - 35 Plummer, G W, and Stabins, J Bleeding duodenal ulcer in infancy. A surgical problem. Report of two cases treated successfully by surgery, J Pediat 37 899 1950
 - 36 Pein N K Neonatal gastrocolic fistula report of cases, Lancet 2 53 1948
 - 37 Buckstein J The duodenal niche—a criterion in the healing of duodenal ulcer, Surg, Gynec & Obst 51 109, 1930
 - 38 Hart, C Über das Ulcus duodeni Med Klin 10 363, 1914
 - 39 Bland Sutton, Sir J On cancer of the duodenum and small intestine, Tr Med Soc London, 38 1, 1915
 - 40 Jefferson, G Carcinoma of the supra papillary duodenum causally associated with pre existing simple ulcer, report of a case and an appendix of 30 collected cases Brit J Surg 4 409, 1916
 - 41 MacCarty, W C Excised duodenal ulcers a report of four hundred and twenty five specimens J A M A 83 1894, 1924

The diagnostic significance of such a finding in the roentgenogram is undeniable, and in a borderline case in which the clinical impression is in doubt, such evidence speaks clearly, in the vast majority of cases, for the perforation of a peptic ulcer.

Exceptionally, the subdiaphragmatic accumulation of gas may be due to its escape from perforations other than those of an ulcer of the stomach or duodenum. Thus Dandy⁸ found such evidence in a case in which operation showed several pin point perforations in a large ulcer of the transverse colon. The entire colon was covered with typhoid ulcers. A similar example of the development of spontaneous pneumoperitoneum was described by Mondor and Porcher.⁹ Also, perforation of an ulcer of the ileum can produce this phenomenon.

I have seen air under the diaphragm from rupture of the ileum in two individuals who attempted to replace a strangulated hernia forcibly, in several other cases as the result of blunt trauma to the abdomen, and in another case as the result of perforation of a diverticulum of the ileum, I have seen it also as the result of perforation of the colon during sigmoidoscopy. Such examples, however, are highly exceptional and, for practical purposes, evidence of spontaneous pneumoperitoneum means the perforation of either a gastric or a duodenal ulcer. The clinical history, such as the absence of evidence of typhoid fever or of ileocolitis or of a gunshot wound, would aid in the elimination of factors other than the perforation of a peptic ulcer as a cause of the free escape of air. While the presence of spontaneous pneumoperitoneum is proof of the perforation of a hollow viscus the reverse does not hold true. That is to say, an acute perforation of an ulcer may take place and yet no recognizable roentgen evidence may be noted. This may be due to the fact that an insufficient amount of air escaped, or that the gastric contents may have been primarily the food of a preceding meal, or that the hole became plugged promptly after perforation.

As a rule, acute perforation of a peptic ulcer demands immediate surgical intervention for closure of the opening. In rare cases, however, the perforation may become walled off by a plastic exudate or the adherence of a pad of omentum, or by a piece of food plugging the hole and preventing undue leakage until reparative processes occur. There may thus be a spontaneous recovery without the aid of the surgeon. In a case of this sort, the original roentgen examination may indicate an accumulation of air under the diaphragm, characteristic of this condition. As the symptoms subside, re-examination may later show a complete absorption of the escaped air. Martin¹⁰ in 1917 described a case of this type in which a male 26 years old, after an attack of agonizing abdominal pain showed the presence of air under the right diaphragm. The patient refused operation. Later he developed an abscess which required drainage. The patient recovered. This was evidently a case of acute perforation of a peptic ulcer with the escape of air but going on to spontaneous closure.

One of the cases of spontaneous pneumoperitoneum described by Copher¹¹ was also apparently of the walled off type of perforation. Roentgen examination showed a deformity of the duodenal bulb, with a local projection of gas which suggested a walled off perforated duodenal ulcer. At the same time, free air was found under both the right and the left diaphragms. There was no escape of the opaque meal into the free abdominal cavity. The case thus had the features of an acute perforation of an ulcer which then became walled off. No operation was performed. This condition was described as the "formes frustes" type of acute perforation by Singer and Vaughan.¹²

DIFFERENTIAL DIAGNOSIS

A rare condition which may simulate the accumulation of air under the right diaphragm is that of interposition of the colon between diaphragm and liver. The differentiation of these two conditions depends

upon the demonstration of haustrations characteristic of the colon. In the event that further doubt exists, a barium enema, by outlining the colon, will show the exact anatomic relationship of the interposed bowel as having been the cause of the translucent subdiaphragmatic shadow. This danger of confusion may arise particularly when an attempt is made to read the freshly developed film in the dark room because of the emergency character of the examination, as occurred in one of the cases reported by Pendergass and Kirk.¹¹

Still another cause from which differentiation may be required is a subdiaphragmatic abscess. In this condition there is a fluid level at the base of the accumulated gas which may undulate on shaking the patient. The demonstration of such a fluid level may be best accomplished by examining the patient with the body erect, in the standing or sitting position, or because of the shift of the fluid level, examination may also be conducted with the patient in the lateral position. A subdiaphragmatic abscess of this kind may, of course, also be a complication of an acute perforation of a peptic ulcer. In some cases, one may note not only the semilunar appearance of the escaped air, but in addition a number of small, walled-off translucent areas, usually along the right lateral margin, representing encapsulated areas of fluid and air. While the subdiaphragmatic abscess is usually on the right side, in rare instances it may involve the left side and actually go on to destruction of the diaphragm.

Acute perforation of an ulcer may occur, in rare instances, after roentgen examination following the administration of barium. In some cases, an unusual degree of manual compression during fluoroscopic observation may perhaps be responsible. However, during a period of many years I can recall only two cases in which the perforation of a peptic ulcer occurred sufficiently soon after roentgen examination to suggest that this may have been an inciting factor. The fact that such a complication occurs so rarely,

considering the large amount of work in alimentary-tract roentgenology carried out at Bellevue Hospital, demonstrates the remarkable safety of roentgen examination in the presence of an active lesion of the stomach or duodenum.

Illustrative Cases An acute perforated ulcer may produce a fistulous tract which may become completely walled off, as illustrated by the case that follows.

T. D., male, aged 45. Three days before his admission to the hospital the patient had a sudden attack of severe, sharp, nonradiating pain, made worse by deep breathing. He had chills and fever and his local physician gave him a hypodermic injection to control his pain. For a period of 6 years until 3 years before admission, he had had recurring attacks of abdominal pain. He then remained symptom-free until this last attack which brought him to the hospital.

Operation disclosed the following. On entering the abdomen a considerable number of dense fibrous and fibrinous adhesions were found, extending between the stomach, duodenum and the liver. Most of the inflammatory process was localized in the lesser omentum or gastrohepatic ligament associated with an inflammatory process in the pars media of the stomach at the lesser curvature. This proved to be an ulcer of the pars media which had apparently ruptured at some previous date and resulted in the formation of a fistulous or sinus tract. No attempt was made to disturb the fistulous tract and the abdomen was closed.

Roentgen examination (Fig. 358 A and B) revealed a fistulous tract extending between the duodenum and the region of the lesser curvature, pars media. Near the lesser curvature of the stomach there was a saccular, ovoid collection of barium. Leading away from this area, the tract became quite narrowed and bifurcated. One part of the tract, irregularly rounded and well circumscribed, extended in a downward direction for a short distance. The rest of the tract, linear in appearance, extended to the right to the region of the duodenal bulb. When the patient was fluoroscoped, the fistulous tract first appeared as if emerging from the region of the duodenal bulb and then finally seemed to extend over towards the large collection of barium in the region of the lesser curvature of the stomach. It therefore appeared as if the fistula had actually originated at the site of the duodenal



FIG 358 (A, *Left*) Ulcer, with walled off fistulous tract (B, *Center*) Same patient (C, *Right*) Same patient Finally disappearance of the fistulous tract was almost complete Only a small rounded collection of barium marks the site of the original tract

bulb and extended to the lesser curvature of the stomach

The surgeon, however, at exploration thought that an ulcer of the lesser curvature was the point of origin

The patient made an uneventful recovery Roentgen examination a few months later (Fig 358 C) showed that the fistulous tract had disappeared except for a very small rounded collection of barium at some distance from the lesser curvature of the stomach

The following case illustrates the presence of a comparatively small amount of air under both the right and the left diaphragm as a result of the perforation of a peptic ulcer

S M male, aged 30 This patient was essentially well until the day of his admission to the hospital when he developed severe crampy, abdominal pains He had imbibed

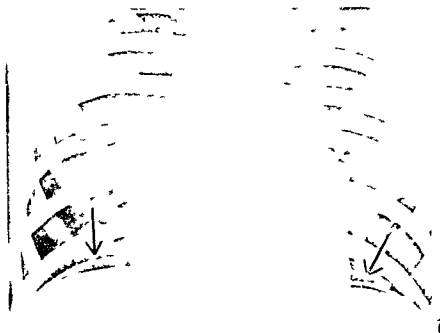


FIG 359 Air under both diaphragms Perforation of duodenal ulcer

considerable alcohol the week before the onset of this pain. He had taken castor oil in the morning because he had not had a bowel movement for 4 days. He was seen by his local doctor, who gave him a hypodermic of morphine, following which he felt relieved. At 5 P. M. that afternoon he had another attack of severe pain in the abdomen. Four years previously he had had a similar episode after lifting a sack of flour to his shoulder. At that time, he was operated on for appendicitis. There was no history of bloody or tarry stools.

Physical examination revealed acute tenderness just below the costal margin to the right of the ensiform cartilage. The abdomen was soft. There was rebound tenderness.

Roentgen examination (Fig. 359) revealed the escape of a small amount of air beneath the right and the left leaves of the diaphragm. It was on the basis of this finding, indicating that he had had an acute perforation, that the patient was operated on. Operation revealed a perforated ulcer on the anterior surface of the duodenum just distal to the pyloric ring.

The significance of the radiologic findings as an evidence of acute perforation of a peptic ulcer is particularly emphasized by this case, since the abdomen was soft on physical examination and the clinical diagnosis was uncertain.

In some cases, the escaped air following perforation of an ulcer may be limited to the right side only, between the right dia-

phragm and the dome of the liver. The escaped air may be huge in amount.

Acute perforation of the stomach may be accompanied by marked evidence of hydro-pneumoperitoneum, as illustrated by the following case.

F. H., male, aged 2 years and 4 months. The child was perfectly well until 2 days before admission to the hospital when there was a sudden onset of vomiting, which was not projectile in character. The vomiting was almost continuous. No blood was present. The abdomen gradually became distended and the respirations grunting in character. Physical examination showed a child in acute distress, cyanotic with grunting respiration. The abdomen was distended and of boardlike rigidity.

Autopsy revealed considerable subcutaneous emphysema of the scrotum, abdominal wall, chest and retroperitoneal tissues. Considerable gas and fluid were present in the abdominal cavity. On opening the stomach, a punched-out, rounded hole through the mucosa was found at about the upper end of the anterior wall of the pars media. The aperture extended through the serosa. On the mucosal side it was about the size of a 5-cent piece.

Roentgen examination (Fig. 360 A and B) showed the enormous amount of escaped air so that the liver and spleen were well outlined. The fluid-level line extended across the entire abdominal cavity. The evidence was

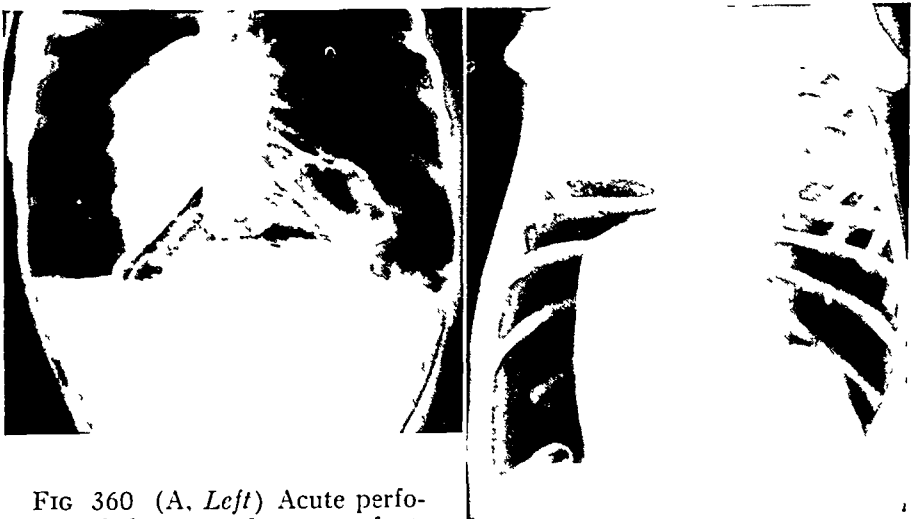


FIG. 360 (A, *Left*) Acute perforation of the stomach in an infant (B, *Right*) Same patient. Note the tremendous amount of air under both diaphragms, outlining the liver and the spleen.

obviously that of an enormous amount of fluid and free air within the abdominal cavity the result of rupture of a hollow viscus

Acute perforation of a gastric ulcer in a newborn is shown in the following case

Baby B, female weight 1920 Gm at birth (normal spontaneous delivery) The infant appeared to be doing well until the fourth day, when she developed rapid progressive abdominal distention. She had been taking her feedings satisfactorily until that morning. The stool evacuations were normal until then. There was no blood in the stool. On physical examination the abdomen was markedly distended and tympanic. No masses were palpable. Rectal examination was negative. There was no blood on the gloved finger.

Roentgenographic examination (Fig 360 C) disclosed a hydropneumoperitoneum indicating the perforation of a hollow viscus with the escape of fluid and gas. Immediate preparations were made for surgical intervention, but the infant died before anything could be done. After death a 20 gauge needle was introduced into the right lower quadrant. Twenty cc of cloudy yellow fluid was removed containing flecks of white curds.

Autopsy revealed perforation of an ulcer near the cardiac portion of the stomach.

Not only may escaped air be recognized when the examination is conducted in the erect position but also with the patient turned from the supine position to his left side (left lateral decubitus position). The film is then placed in front of the abdomen and the ray directed postero-anteriorly. The air will then be detected between the liver and the right lateral abdominal wall. Not only is the evidence obtained satisfactory, but examination by this procedure is as a rule easier for the patient. Figure 361 shows the findings in a case in which the surgeon at operation discovered a perforated prepyloric ulcer (M. M., aged 44).

Of even greater importance than the fact that the left lateral position may be less disturbing to the patient particularly when he is in shock is the fact that small amounts of air may thereby be discovered which might otherwise entirely escape detection.



FIG 360 C Acute perforation of a gastric ulcer in a newborn. Examination in the erect lateral position. Note the fluid level extending across the entire abdominal cavity capped by an enormous amount of gas. This had resulted from the escape of fluid and air from the stomach through the perforated gastric ulcer.

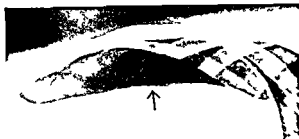


FIG 361 Acute perforation of a prepyloric ulcer. Note the escaped air in the left lateral decubitus position.

J B, male, aged 65 The patient gave a 15-year history of recurrent attacks of epigastric distress About 1 hour before his admission to the hospital, he developed severe epigastric pain "that doubled him up" Physical examination of the abdomen revealed generalized tenderness and marked rigidity Operation disclosed a perforated ulcer on the anterior wall of the stomach

The preoperative roentgen examination showed no free air under the right diaphragm with the patient in the erect position (Fig 362 A) In the left lateral position, however, there was a very small but definite collection of air between the liver and diaphragm at the right costal margin (Fig 362 B) Examination in the left lateral position for the demonstration of escaped air is therefore of value for two reasons (1) Even if the air can be readily demonstrated with the patient in the erect position, it may be much easier at times for the patient to turn on his left side for radiography (2) The left lateral position may also be advantageous, since a small amount of escaped air demonstrable in this position may escape detection when the patient is upright

Following acute perforation, the ulcer may become walled off and the patient may make an uneventful recovery without operation This is illustrated by the following case, in which operation confirmed the presence of an ulcer, the perforation in which had become sealed

M. A, male, aged 49 This patient had epigastric distress of 10 years' duration The

pain occurred about 1 hour after meals and was temporarily relieved by liquid food No radiation of pain was present Brief periods of several months' duration occurred during which there was freedom from distress Vomiting occurred occasionally, with no blood present, until 3 weeks prior to his admission to the hospital, when he vomited dark blood No tarry stools were noted Two weeks later he had a sudden acute attack of abdominal pain of great severity, not relieved by liquid food as heretofore The pain subsided the following day At the time of admission he complained of discomfort across the upper abdomen The most important finding on physical examination at this time was obliteration of liver dullness The abdomen was soft

Roentgenographic examination revealed the presence of pneumoperitoneum, particularly indicated by the presence of air between the dome of the liver and the diaphragm, as noted by the arrows in the accompanying roentgenogram (Fig 363) This accounted for the obliteration of liver dullness Because of the presence of air in the abdominal cavity, a diagnosis of perforated ulcer was made

At operation a moderate amount of clear straw-colored fluid was found above and below the liver On the lesser curvature of the pars media an ulcer was found, with peritoneal involvement The perforation was completely sealed Gastro-enterostomy was performed

An excellent illustration of the "formes frustes" type of perforation of a peptic ulcer with spontaneous recovery is to be seen in the next case



FIG 362 (A, *Left*) Perforation of a gastric ulcer Examination in the erect position Note the absence of air under the right diaphragm (B, *Right*) Same patient Examination in left lateral decubitus position Note the small amount of escaped air between the liver and the right lateral border of the abdominal wall

S F, male, aged 59 About 7 years previously this patient had developed "stomach pain" following eating. He remained well for 5 years, when he had a recurrence of his gastric symptoms. Distress occurred immediately after eating and was relieved by soda. This would be followed 3 hours later by vomiting. He had lost 10 pounds in the 6 months preceding his admission to the hospital. At the time of hospitalization he had a severe attack of abdominal pain, which caused him to double up. The pain was generalized, sharp and radiated to the back. During the preceding 2 or 3 days he had vomited black material. He had not noted blood in the stool.

Physical examination revealed abdominal rigidity. There was moderate tenderness throughout the abdomen. Rebound tenderness was present, but no evidence of obliteration of liver dullness. Clinically the patient was considered to have an acute surgical abdomen.

Roentgen examination at this time (Fig 364 A) revealed definite evidence of air under both diaphragms. The diagnosis was acute perforation, probably of a peptic ulcer. The patient refused operation, in spite of which he made an uneventful recovery.

A second roentgen examination was made 10 days later (Fig 364 B). There was now no

evidence of any air under the diaphragm, the air presumably had been absorbed. A roentgen study of the stomach and the duodenum showed a definite deformity of the duodenal bulb which was characteristic of ulcer.

Both from the clinical standpoint and from the roentgen evidence, there can be no doubt that the patient had an acute perforation of a duodenal ulcer with the escape of air under both diaphragms. There was apparently a complete closure of the duodenal ulcer, the presence of which was later demonstrated roentgenographically. The air under the diaphragm was completely absorbed within 10 days.

The gradual absorption of escaped air after perforation of a peptic ulcer may be noted in the following case.

J Z, male, aged 37 Three years before his admission to the hospital the patient had had a sudden attack of abdominal pain with out any previous symptoms and an emergency closure of a perforated peptic ulcer was done at the hospital. From then on he had been attending the out patient ulcer clinic. The night before admission he again had a sudden attack of severe abdominal pain which brought him to the hospital. Physical exam

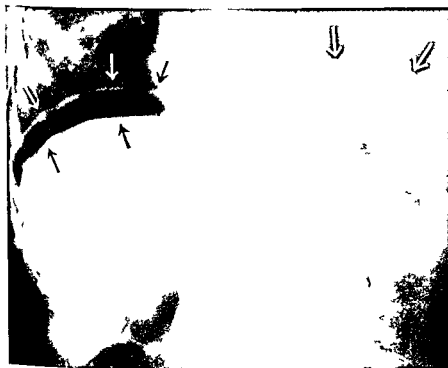


FIG 363 Air under both diaphragms due to perforation of a gastric ulcer which became completely sealed off

ination revealed a rigid abdominal wall with excruciating tenderness in the left upper quadrant

Within a few hours after his arrival at the hospital, the abdominal wall had become soft and the pain had subsided. The patient made an uneventful clinical recovery without surgical intervention.

Roentgen examination (Fig 365 A) showed the appearance of the lesion on the day the patient was admitted to the hospital. Air was present under both diaphragms, particularly under the right, the result of a perforated hollow viscus.

Seven days later, examination showed a marked diminution in the amount of escaped air (Fig 365 B). There was none under the left diaphragm and very little under the right diaphragm. Three days thereafter (Fig 365 C) there was no air under either diaphragm. Within 10 days, therefore, all evidence of escaped air had disappeared. Two days later, roentgen examination of the stom-

ach showed a small cone-shaped niche of the lesser curvature, pars media.

The evidence, then, was that of a gastric ulcer that had perforated, with the escape of air lodging under both diaphragms. The perforation was sealed off and the escaped air was completely absorbed within 10 days.

The following is a completely documented story of a gastric ulcer which had perforated with the escape of air into the abdominal cavity with recovery and later resection of the lesion.

The patient C. C., male, aged 63, gave a history of recurrent episodes of epigastric pain during a period of 17 years. Each episode lasted about 4 weeks, following which he would remain well for about 2 years or so. He was relieved on an ulcer regimen. On the day of admission to the hospital he complained of abdominal pain, vomiting and constipation. Immediately on being admitted 600

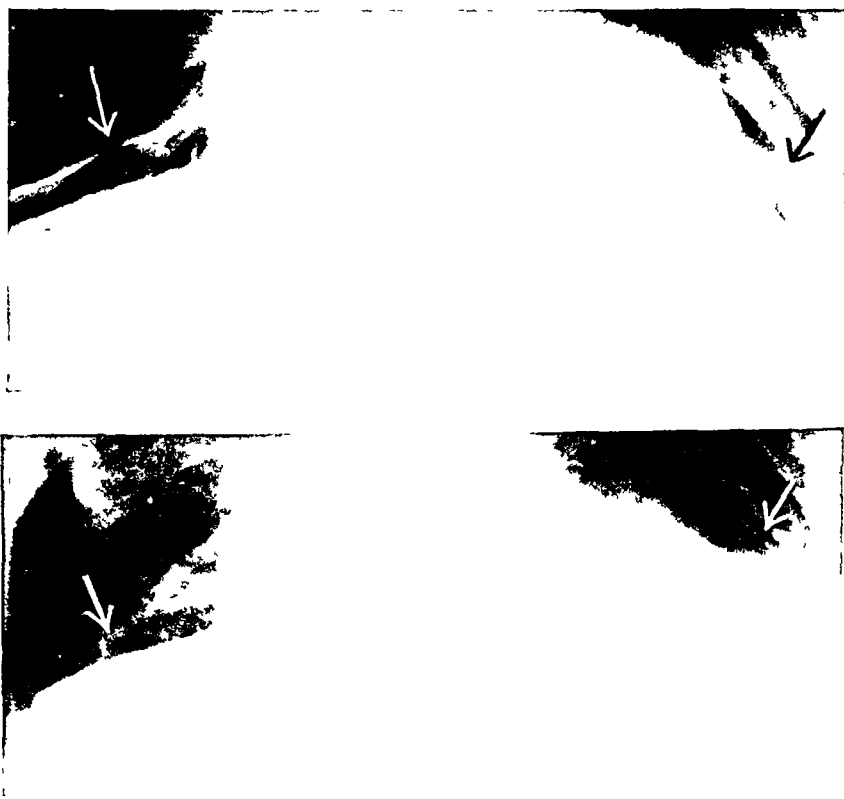


FIG 364 (A, Top) Air under both diaphragms following perforation of a duodenal ulcer (B, Bottom) Same patient 10 days later, showing the complete disappearance of air under both diaphragms. There was no operative interference.

cc of brown and blood stained fluid was aspirated through a Levin tube shortly after he had vomited approximately 500 cc of similar fluid

Physical examination of the abdomen revealed tenderness in the epigastrium marked voluntary spasm of the upper half of the rectus muscles and slight rebound tenderness. Roentgenographic examination of the chest

FIG 365 (A *Top*) Note air under the right diaphragm. The patient was not operated on. (B, *Center*) Same patient. Examination 7 days later. Note the marked diminution in the amount of air under the right diaphragm. (C, *Bottom*) Same patient 3 days later and 10 days after (A). Note the complete disappearance of air under the diaphragm.

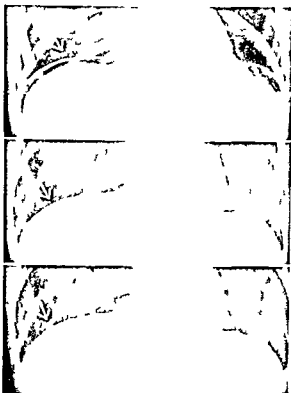


FIG 366 (A *Top left*) Air under the right diaphragm at the time of the acute perforation of the gastric ulcer. The patient was treated conservatively. (B *Top right*) Appearance 4 days later. Note the almost complete disappearance of the air under the right diaphragm. (C *Center left*) Examination 16 days after (B) or 20 days after (A). Note the complete disappearance of the escaped air at this time. (D, *Bottom left*) Roentgenographic appearance showing the walled off perforated ulcer adherent distally. (E *Center right*) Appearance of the resected specimen. The floor of the ulcer had been completely destroyed and it was sealed off by adherence to the duodenum from which it was stripped at the time of the resection.

and scout films of the abdominal cavity revealed air under the right diaphragm. The loops of small intestine were abnormally dilated. However, the bowel sounds were active, and he passed gas per rectum. The clinical diagnosis was perforation of a peptic ulcer. He was treated conservatively with gastric suction, the administration of antibiotics and intravenous fluids. He made a satisfactory recovery.

Figure 366 A taken on the day of admission to the hospital shows the air under the right diaphragm. Figure 366 B, 4 days later shows only a minimal amount of air under the right diaphragm. Figure 366 C, 16 days later shows complete disappearance of the air under the right diaphragm. In this way it was possible to trace roentgenographically the improvement which took place clinically. The day after the completion of the last study recording the total disappearance of the escaped air a roentgenographic examination of the stomach was made. The examination revealed evidence of a sealed-off perforating ulcerating lesion of the lesser curvature of the stomach at about the junction of the pars pylorus and the pars media, apparently adherent to the stomach at its distal portion. There was some obstruction at the pyloric outlet, with very little of the barium escaping

into the duodenum (Fig 366 D). A subtotal gastrectomy was done. Figure 366 E shows the area of ulceration in the resected specimen. The floor of the ulcer had been completely destroyed and sealed off by adherence to the duodenum from which it was removed.

Another result of the acute perforation of a peptic ulcer is the development of a subphrenic abscess, as illustrated by the next case.

J. M., male, aged 35. In the preceding 2 years this patient had been having frequent attacks of crampy abdominal pain associated with vomiting. Two weeks before his admission to the hospital this pain had become almost constant. On the morning of his admission he experienced a knifelike pain in the epigastrium, which doubled him up.

Physical examination revealed boardlike abdominal rigidity. The tenderness was generalized throughout the abdomen but was more marked in the area midway between the umbilicus and the xiphoid process.

At operation, the parietal peritoneum was acutely inflamed. Free fluid was present throughout the peritoneal cavity. A perforation of a chronic ulcer, situated on the anterior wall of the duodenum, was present,



FIG 367 Subphrenic abscess secondary to perforation of a duodenal ulcer. Note the upward dislocation of the right diaphragm and the fluid level capped by gas.

with marked inflammatory reaction about it. The intestinal wall about the perforation was thickened and friable. The perforation was sutured and the abdomen drained. Following this, the patient had a stormy course, with septic temperature and a low leukocyte count. A second operation was performed about 6 weeks later. At this time, a well walled off subphrenic abscess was found just over the liver, containing approximately 1 quart of thick, green, foul smelling pus.

Roentgen examination prior to the second operation (Fig 367) revealed marked elevation of the right dome of the diaphragm. Below the diaphragm was an elliptical gas space with a fluid level indicating the presence of a subphrenic abscess.

Ordinarily, when perforation of a duodenal ulcer gives rise to a subphrenic abscess, this abscess is located on the right side. The next case is unusual in that the abscess developed in relation to the left diaphragm.

F. L., male, aged 56. The patient had been suffering from a duodenal ulcer for about 2 years. There had been evidence of hemorrhage on several occasions, with coffee ground vomitus and tarry stools. Five weeks prior to his admission to the hospital after drinking

to excess he developed a sudden, severe hemorrhage with epigastric pain. His temperature rose to almost 102°F . He had lost 40 pounds.

Physical examination revealed the presence of a visible and palpable mass, nodular and tender, and located in the left upper quadrant; it was tympanitic to percussion. The patient was losing blood faster than it could be replaced by transfusion and death ensued.

Roentgen examination a few days prior to his death (Fig 368) revealed a circumscribed accumulation of gas in the left subphrenic region. The floor of this cavity was formed by the spleen and the greater curvature of the stomach. In addition, there was a deformity of the duodenal bulb. No air was demonstrable under the right diaphragm.

On opening the upper third of the abdomen at autopsy a considerable quantity of gas escaped. Examination of the abdomen revealed a large, walled off cavity in the left upper quadrant, containing considerable mucus and blood which was very dark and was not clotted. The boundaries of this cavity consisted of the floor, formed by the left lobe of the liver, the stomach and the spleen. Superiorly, the peritoneum was firmly adherent to the diaphragm and, on palpation the diaphragm was found to have been eroded through. Anteriorly, it was bounded by



FIG 368 Left subphrenic abscess secondary to perforation of a duodenal ulcer

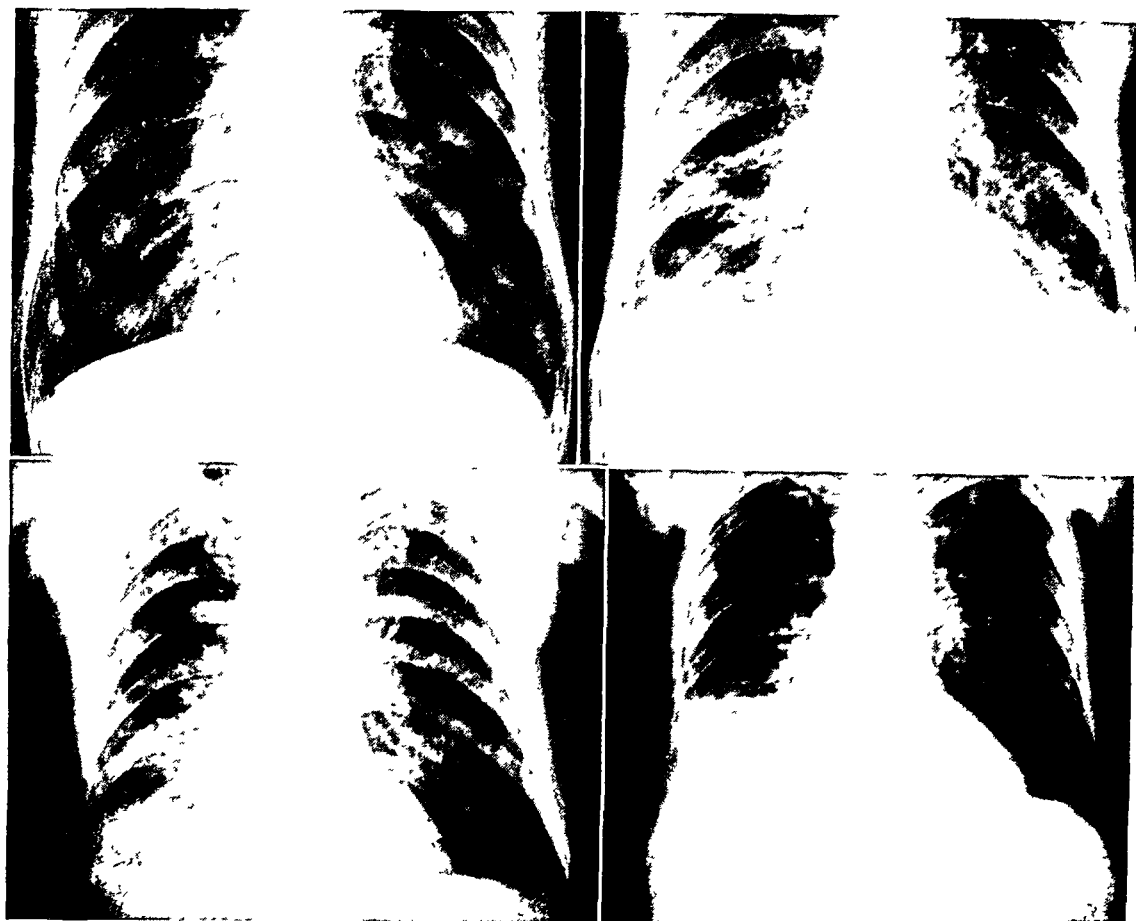


FIG 369 Evolution of a subdiaphragmatic abscess after gastric resection for carcinoma (A, *Top, left*) Normal appearance of the area of the right diaphragm before operation (B, *Top, right*) Examination 6 days after operation Note very small subdiaphragmatic collection of gas (C, *Bottom, left*) Examination 4 days later (10 days after operation) At this time note (1) the elevation of the right diaphragm, (2) the right basilar pneumonitis, (3) the small gas-capped subdiaphragmatic fluid level (D, *Bottom, right*) Four days later (14 days after operation) examination shows an exaggeration of the gas-capped fluid level and an increase in the basilar pneumonitis obscuring the right diaphragm The subdiaphragmatic abscess was confirmed at operation and autopsy

peritoneum and greater omentum, and inferiorly, by greater omentum and transverse colon. There was no generalized involvement of the abdomen, the cavity being completely walled off by the boundaries already mentioned. The stomach was normal. On the superior surface of the duodenum just beyond the pylorus there was a perforation, about 1 cm in diameter, communicating with the cavity previously described. The diagnosis was perforated ulcer of the duodenum, with subphrenic abscess on the left side.

The gradual evolution of a subphrenic abscess is illustrated by the following case:

A P, male, aged 68. After a subtotal gastrectomy for carcinoma of the stomach the patient continued to run a spiking fever. The preoperative roentgenographic examination of the chest shows a normal right diaphragm (Fig 369 A). Examination 6 days after operation (Fig 369 B) shows a minimal amount of gas beneath the right diaphragm. Four days later (Fig 369 C) examination shows the following: (1) upward elevation of the right diaphragm, (2) a small localized area of gas beneath the diaphragm, (3) pneumonitis at the base of the right lung. Figure 369 D, taken 4 days after Figure 369 C and 8 days after Figure 369 B, shows a further spread of the

pathologic process The encapsulated collection of gas has increased, and the fluid level is much wider than in Figure 369 C There is also an increase of the pneumonitis at the right base partially obscuring the outline of the diaphragm In this manner it was possible to trace the definite development of classical features of a subdiaphragmatic abscess

At operation there was "a large subphrenic abscess of the posterior superior space over the dome of the liver The abscess contained at least 1,500 cc of foul smelling fluid pus The abscess cavity was well walled off The abscess cavity was aspirated then opened All the pus was evacuated Two large rubber tubes were inserted into the abscess cavity"

At autopsy there was a large abscess cavity extending above the right dome of the liver The right kidney formed the floor of the abscess cavity

EFFECTS OF OPERATION ON PERFORATED DUODENAL ULCER

In the case of suture of a perforated duodenal ulcer, the duodenal bulb occasionally

may show no evidence of any deformity Frequently, however, a considerable degree of duodenal deformity is present The question is often asked whether there has been a recurrence of the ulcer If only deformity of contour is present, that question cannot be determined by roentgen examination alone unless a niche can be shown A persistent duodenal deformity may indicate *only the scar of the original ulcer, or the deformity, to some degree, at least may have been produced by the operative interference*

In the suturing of a duodenal perforation, inversions of the cut edges may diminish the lumen of the duodenal bulb, particularly in those cases in which considerable narrowing has already resulted from previous cicatrization produced by the ulcer These mucosal swellings which project within the lumen as a result of suturing may gradually diminish, however, as the inflammation subsides

REFERENCES

- McPeak, C N Benign duodenocolic fistula with report of two cases, *Radiology* 34 343 1940
- McClinton, J B Nonmalignant duodenocolic fistula, *Canad M A J* 51 434 1944
- Somogyi, Gyula Röntgendiagnose eines retroperitoneal perforierten Zwölffingerdarmgeschwurs *Fortschr Geb Röntgenstrahlen* 63 221, 1941
- King, J D Spontaneous renoduodenal fistulas, *Radiology* 54 82 1950
- Popper, H Die Diagnose der Darmperforation mit Hilfe der Röntgendurchleuchtung, *Deutsche med Wchnschr* 41 1034 1915
- Weiland, W Ein röntgenologisches Phänomen bei perforiertem Magengeschwür *München med Wchnschr* 62 537 1915
- Lenk, R Röntgenbefunde bei frischen Bauchschüssen *München med Wchnschr* 63 1278, 1916
- Dandy, W E Pneumoperitoneum a method of detecting intestinal perforation an aid in abdominal diagnosis *Ann Surg* 70 378, 1919
- Mondor, H and Porcher, P Examens radiologiques d'urgence des péritonites par perforation du tube digestif, *J chir* 41 20 1933
- Martin, W Subacute duodenal perforation *Ann Surg* 65 773 1917
- Copher, G H Demonstration of spontaneous pneumoperitoneum by the roentgen ray an aid in the diagnosis of acute perforating peptic ulcer *J A M A* 82 781, 1924
- Singer, H A and Vaughan, R The formes frustes type of perforated peptic ulcer *Surg Gynec & Obst* 50 10 1930
- Pendergrass, E P and Kirk, E Significance of gas under the right dome of the diaphragm with discussion of hepatoptosis *Am J Roentgenol* 22 238, 1929

The Duodenal Curve

VARIATIONS IN SHAPE

The duodenal curve may show considerable variation in shape. To determine these differences Dwight¹ recorded his observations on the anatomy of the duodenum in a series of 54 adult bodies. His procedure was as follows: He inflated the duodenum before disturbing the intestines and took note of its shape and position. He then made a cast of the duodenum in mineral wax. The primitive form of the duodenum is that of a ring not quite complete. The U shape and V shape were the most common forms. Other less common forms were ring-shaped and C-shaped. In 10 cases the shape was indeterminate and represented mixtures of the U, C and ring shapes. Five duodenum were absolutely irregular. In 26 cases the duodenum was to the right of the aorta until just before the terminal flexure. It was entirely to the right of the aorta in 6 cases.

There is also considerable variation in the form of the duodenojejunal flexure², as pointed out by Harman. In an analysis of a number of cases showing such variations, he found that, in 4 cases, the direction of the bend was to the left in a bold curve and then downward. In 6, the bend was forward and straight downward. In 1 case, the direction was forward and then downward, turning slightly to the right. In 2 cases, the bend was forward and then transversely toward the right side.

In the usual type of curve the duodenojejunal junction may be noted projecting posteriorly and above the lesser curvature

of the stomach. A knuckle of barium in this area in some circumstances may momentarily simulate the niche of a gastric ulcer during fluoroscopic examination or on a single film. Careful fluoroscopic survey will show the inconstancy of this finding and the continuity with the rest of the duodenojejunal curve.

THE FUNCTIONAL BEHAVIOR OF THE DUODENUM BEYOND THE BULB

Although the duodenal bulb itself is of smooth outline and its mucosal folds, when present, run in its longitudinal axis and bear a strong similarity to the rugal folds of the stomach, the mucosal relief of the duodenum beyond the bulb shows the presence of transverse folds producing an irregularity in the contour of this region. This abrupt change is due to the beginning of the formation of the valvulae conniventes. Peristaltic activity ordinarily forces the contents distally in a rapid manner so that it is not always easy to obtain a complete delineation of a homogeneously filled duodenal curve.

There are, in addition, some interesting peculiarities in the functional behavior of this region. Reverse duodenal peristalsis may occasionally be noted, with typical to-and-fro, writhing movements, frequently forcing the barium back toward the pylorus. In many cases this is probably a purely physiologic phenomenon, its purpose being to produce a more intimate admixture of the duodenal contents with the biliary

and pancreatic secretions. Only when there is definite disturbance of motor function and abnormal delay in the emptying of the duodenum may one justifiably consider the presence of an abnormal factor as the cause. An interesting light was thrown upon this type of behavior by the work of Ochsner.^{2,4} He made an anatomic study of the duodenum in a number of cadavers and stated that in all the specimens examined there was a more or less marked thickening of the intestinal wall, usually from 3 to 10 cm. below the entrance of the common duct, due to a considerable increase in the circular muscle fibers. The findings suggested to Ochsner the presence of a sphincter, with the physiologic function of causing a retention of the contents in the upper portion of the duodenum for a sufficiently long period to permit of an intimate admixture with the biliary and pancreatic secretions. In some cases, two regions of hypertrophy of the circular muscle of the duodenum were demonstrated. In pathologic states Ochsner assumed that there might be a hyperfunction of this sphincteric area with dilatation of the proximal portion of the duodenum.

These findings by Ochsner were only partially confirmed by Boothby, who studied the duodenums in twelve autopsies. He found certain irregularities of thickness in the musculature of the duodenum; these, however, were insignificant as compared with those of a true sphincter such as the pylorus. He found undoubted evidence of moderate variations in the thickness of the muscle of the duodenum.

A more important factor clinically in the production of reverse duodenal peristalsis is the pressure to which the transverse duodenum is subjected when it is compressed by the root of the mesentery crossing it anteriorly against the vertebral column posteriorly. This was shown in the wax casts of the duodenum prepared by Thomas Dwight. In many of these there was an impression on the cast at the site where the transverse portion is crossed by the root of the mesentery. In addition there

was evidence of the pressure produced on the posterior wall of the wax cast by the aorta and the vertebral column. In some instances there was considerable dilatation of the duodenum proximal to the region of constriction. Such compression of the duodenum is exaggerated by the prolapse of the loop of small intestine increasing the pressure produced by the root of the mesentery. The pull of the cecum, the hepatic flexure or the transverse colon may similarly lead to compression of the duodenum. Constriction of the duodenum by the root of the mesentery is a cause of duodenal dilatation was described at laparotomy by Bloodgood.

Another potential cause of transient obstruction of the duodenum is angulation of the duodenojejunal junction which may be enhanced by the activity of the suspensory muscle of the duodenum. Haley and Perry studied the duodenal ligament in 64 cadavers. In 57 or 59 per cent either a muscle or a ligament could be definitely demonstrated while in 7 or 11 per cent neither could be found. It was attached to various parts of the second, third and the fourth portions of the duodenum as well as the duodenojejunal junction. In the largest number of cases (14) it was attached to the flexure as well as the third and the fourth parts of the duodenum. Muscle fibers were present. In only 5 or 8 per cent of the specimens in which a definite anatomic structure could be demonstrated was it attached only to the flexure. The suspensory ligament containing smooth muscle when attached to the duodenojejunal flexure may by contraction contribute to obstruction by exaggerating the degree of angulation.

A number of other factors may be responsible for the within duodenum associated with reverse peristalsis. The duodenal curve passing in close relation to a peptic ulcer may become partially enmeshed in the inflammatory process and lead to various degrees of obstruction. In carcinoma of the stomach obstruction of the duodenum may arise in two ways: (1) metastatic

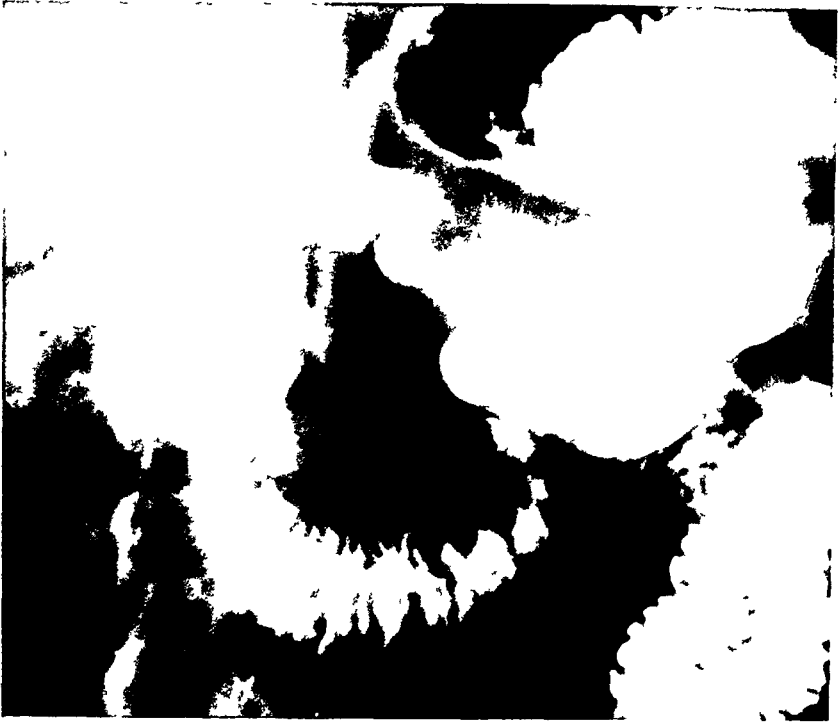


FIG 370 Normal duodenal curve. The apparent enlargement of the duodenal curve is due to the fact that the examination was made with the patient in the prone position causing upward displacement of the stomach

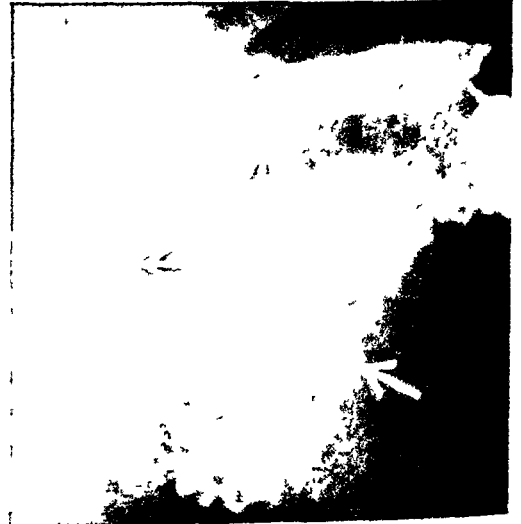
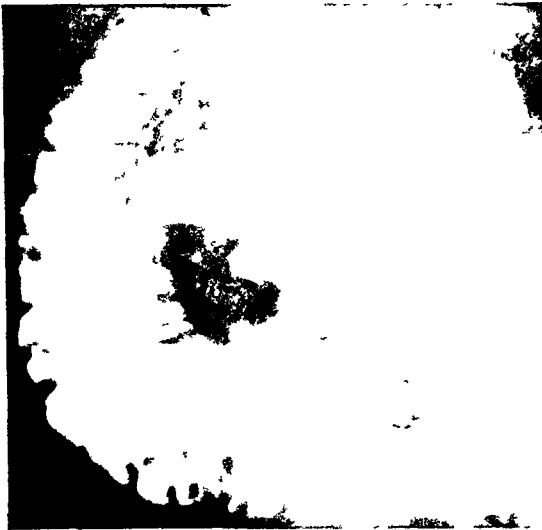


FIG 371 (Left) Normal duodenal curve

FIG 372 (Right) Normal duodenal curve. Note the transition from longitudinal folds in the proximal portion of the duodenum to the broad transverse folds of the midportion and to the fine mucosal structure of the distal portion



FIG 373 Mucosal appearance of the distal portion of the duodenum



FIG 374 Alterations in the appearance of the duodenal curve due to peristaltic activity



FIG 375 Papilla of Vater?

retroperitoneal nodes may cause compression, and (2) direct extension of the primary growth may press upon various portions of the duodenal curve. Tumor of the pancreas, a retroperitoneal tumor, and adhesions may directly obstruct the duodenum.

Intrinsic new growth of the duodenum as a cause of obstruction will be discussed in a later chapter dealing with this subject.

Illustrative Cases Examples of the normal sweep of the duodenal curve are illustrated in Figures 370, 371, 372.

Figure 372 shows the transition from the longitudinal folds in the proximal portion of the duodenum to the broad transverse folds of the midportion and, in the third portion of the duodenum to a pattern of fine interlacing folds similar to that which may be noted in the jejunum. The detailed appearance of the distal portion of the duodenum may be noted in Figure 373. As the result of peristaltic activity longitudinal folds may appear in various portions of the duodenal curve (Fig. 374).

At times, a small, rounded translucent area may be noted within the confines of the second portion of the duodenum, which is probably due to the papilla of Vater (Fig. 375). In the first place, it corresponds anatomically to the location of the papilla. Secondly, tumors of the papilla of Vater corroborated by surgery or autopsy may produce roentgen evidence of deformity in this region.

In addition to the rounded translucent area within the confines of the second portion of the duodenum produced by the papilla of Vater, sometimes it may also be possible to show a fleck of barium apparently within the ampulla (Fig. 376).

An example of the roentgen findings in delay in the passage of the barium beyond the transverse portion of the duodenum is furnished by Figure 377. Note that the duodenal curve is essentially normal as to position. At the junction of the second and third portions of the duodenal curve, there are stasis and dilatation. These findings

were persistently present in films taken at different intervals, although the stomach and duodenum had otherwise altered their appearance as the result of physiologic activity. The common cause of this condition, when marked, is apparently pressure of the root of the mesentery as it traverses the transverse duodenum.

Fluoroscopically, such stasis and dilatation in the proximal portion of the transverse duodenum are usually associated with reverse peristalsis and the characteristic to-and-fro "writhing" movement of the duodenum proximal to the area of obstruction.

CONGENITAL ANOMALIES OF THE DUODENUM

The most common type of congenital anomaly of the duodenum is that due to variations in position. Instead of the normal duodenal curve, almost every variation in its course may be noted. A common congenital disorder under this classification is redundancy of the second portion of the duodenum just distal to the duodenal bulb. This area, filled with barium, may simulate a diverticulum when the proximal and the distal arms of the redundant area overlap. In order to differentiate between the two conditions, careful observation is essential during fluoroscopic examination. One may then note the course of the barium through the redundancy before overdistention occurs. Moreover, one may be able to separate the loops manually. Also, films taken in different positions may help to show the true course of the barium through the duodenal loop and eliminate the suspicion of a diverticulum.

The duodenum may show failure of rotation as part of the congenital anomaly of nonrotation of the colon. As a result, the second portion of the duodenum, instead of turning downward and to the left, descends directly to become continuous with the jejunum, to the right of the midline. There is thus an absence of the usual third and

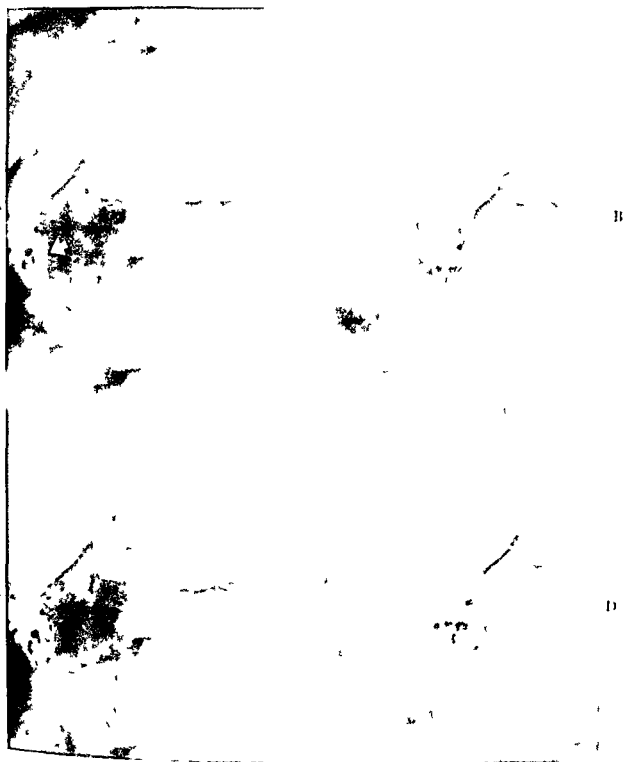


FIG. 316. Translucent area apparently produced by the papilla of Vater with a central fleck which may represent barium within the ampulla. Note the persistence of the finding in the different views. A, B, C, and D. One cannot rule out the possibility of an aberrant pancreatic nodule with a central umbilication containing a fleck of barium.

fourth portions of the duodenum and of the duodenojejunal junction at the ligament of Treitz. Persistence of the duodenal mesentery beyond embryonal life, observed particularly in association with nonrotation of the duodenum may permit an unusual degree of mobility ("duodenum mobile")⁸

Almost every anomalous variation in the course of the duodenal curve has been described. It may be triangular or completely circular, the duodenojejunal junction approaching the bulb. The third portion of the duodenum, instead of extending to the left, may turn to the right, extend upward along the lateral border of the second portion, and at varying levels turn to the left, crossing it in such manner as to form at times a figure 8. It may pass anteriorly or posteriorly to the second portion. Or the third portion, ascending along the medial aspect of the second portion, may fail to cross the vertebral column. The jejunum, which ordinarily turns to the left of the duodenojejunal junction, may be directed inward toward the midline.

These variations in the configuration of the duodenal curve ordinarily appear to have no clinical significance. The only time one might be justified in attributing significance to such anomalies would be if they were associated with disturbed motility and stasis of the barium within the loops.

A point of some significance is the fact that the duodenal curve, therefore, does not always encircle the head of the pancreas. This is one reason why enlargement of the duodenal curve is not a necessary concomitant in all cases of tumor of the pancreas.

Even when the course of the duodenal curve is in the usual direction, the size of the duodenal curve may show considerable variation within normal limits. Moreover, the duodenal curve may vary in apparent size in different individuals because of the position of the stomach. In the hyposthenic, with a low position of the stomach, the duodenal curve may appear smaller than in the hypersthenic individual, with a transverse position of the stomach. In addition, the



FIG. 377 Note the delay at the midportion of the transverse duodenum, with puddling of the barium at the junction of the second and the third portions.

duodenal curve will vary in apparent size with a change in the position of the patient from the erect to the prone. When lying down, the stomach, being displaced upward to a greater extent than the duodenum, causes the duodenal curve to appear to be of increased size. This point is of particular importance in the interpretation of the significance of abnormal enlargements secondary to tumors of the pancreas.

Another anomaly in the position of the proximal duodenum is that resulting from fixation at the junction of the first and second portions of the duodenum by congenital bands. Angulation may in some cases be only apparent, however, and due to the direction of the roentgen rays. By turning the patient through various degrees of obliquity it may be possible to show that the contour is actually curved. Congenital bands may similarly produce variations in the contour of the duodenal curve in other regions, particularly in the descending portion, with the production of angulations.

Considerable clinical importance has been attributed to angulations produced by congenital bands.^{9, 10, 11} The significance of such evidence must be carefully evaluated, however, in the light of the clinical history and the presence of definite derangement of function. If the barium flows through the apparently distorted duodenal curve without visible evidence of impairment, there is probably little likelihood that the congenital anomaly is an important factor in the explanation of the symptoms. In some cases distortion in the configuration of the duodenal curve may be the result of acquired adhesions, secondary to operative procedures.

In addition to apparent fixation of the junction of the first and second portions of the duodenum by periduodenal bands, there is a normal fixation by means of the hepato-duodenal ligament. When the stomach becomes ptosed, particularly as the result of enlargement and dilatation, a pull may be exerted upon the fixed portion, which

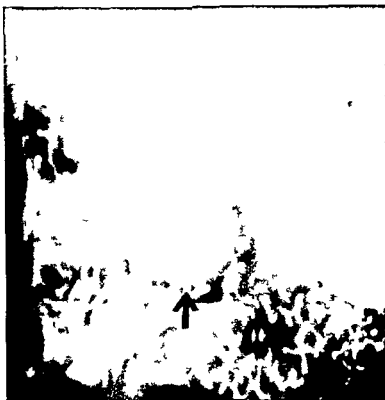


FIG 378 Redundant second portion of the duodenum



FIG 379 Anomaly of the duodenal curve



FIG 380 Anomaly of the duodenal curve. Note the figure-8 appearance

cannot partake, at least to the same degree, in the downward gastric displacement. As a result, the pyloroduodenal arm assumes a vertical direction with the patient examined in the erect position. There is thus an increase in the distance between the lowest point of the greater curvature and the position of the duodenal bulb. This may offer an additional mechanical burden to an already ptosed stomach and may greatly increase the difficulty of gastric evacuation.

In a similar manner, fixation of the duodenojejunal junction may possibly explain the difficulty in duodenal emptying encountered in some cases.

Because of the rapid emptying of the duodenal curve, it is often difficult to visualize this region in its entirety for detailed study. In such cases, the duodenogram may be employed for the complete and isolated visualization of this region.¹² According to this procedure, an intestinal tube is passed by mouth until the distal end reaches the jejunum. The last 8 or 10 inches of the tube are perforated. About 20 cc of barium sus-

penston, injected through the oral end by means of a syringe simultaneously escape through these perforations, thereby filling the entire duodenal curve in an isolated and homogeneous manner. Films are made promptly on completion of the injection.

Illustrative Cases Redundancy of the second portion of the duodenum just distal to the bulb is illustrated by Figure 378. One may readily see how overlapping of the proximal and distal arms of this redundant portion might produce an appearance simulating that of a diverticulum. Occasionally, also, the second portion of the duodenum, under these circumstances, may angulate so sharply with the bulb as to create the impression of a smooth defect of the greater curvature of the bulb itself and thus lead to an erroneous suspicion of ulcer. This is one reason among others why it is important to visualize the entire duodenal curve in addition to the bulb itself.

An example of an uncommon type of congenital anomaly in the course of the duodenal curve is illustrated in Figure 379. The second portion of the duodenum, instead of completing its curve by crossing to the left

to join the duodenojejunal junction, turns laterally upward and then crosses to the left at a level above the duodenal bulb itself. In such cases, obviously, the head of the pancreas does not "sit in the lap of the duodenal curve." Such anomalies may be devoid of any clinical significance.

Another unusual anomaly of the duodenal curve is shown in Figure 380. Note the figure 8 loop produced by the duodenal curve.

In Figure 381 the second portion of the duodenum finally turns to the right, curling on itself and then joining the third portion of the duodenum running transversely to the left. In Figure 382 the second portion of the duodenum joins the third portion, which ascends lateral to it, then joining the jejunum, which then continues downward and to the right.

An interesting type of congenital anomaly of the duodenal curve is that associated with a failure of rotation of the loops of small intestine to the left. In such cases the second portion of the duodenum extends to the right, leading directly into the jejunum, which is also present on the right.



FIG. 381 Anomaly of the duodenal curve



FIG 382 Anomaly of the duodenal curve

side (Fig 383) In cases of this kind, the colon also shows a concomitant failure of rotation to the right, and on examination is found present on the left side of the

abdomen. The finding of a congenital anomaly of this nature in the duodenal curve should call one's attention to the fact that it is associated with a failure of rotation of the small and large intestine The appearance of nonrotation of the colon will be considered in a later chapter

Angulation at the junction of the first and second portions of the duodenum by postoperative adhesions is illustrated by the following case

F B , male, aged 79 The patient came to the hospital complaining of severe abdominal pain of 4 hours' duration He had had a cholecystostomy 33 years previously Physical examination of the abdomen was negative except for the scar of the previous operation On exploration, many dense postoperative adhesions were found fixing the pylorus and duodenum to the anterior abdominal wall The gallbladder was found pressing down on the superior margin of the duodenum

Roentgen examination (Fig 384) revealed a smooth defect on the greater curvature border of the duodenal bulb as well as in the second portion of the duodenum just distal to the bulb There was an angulation at its junction with the rest of the second portion

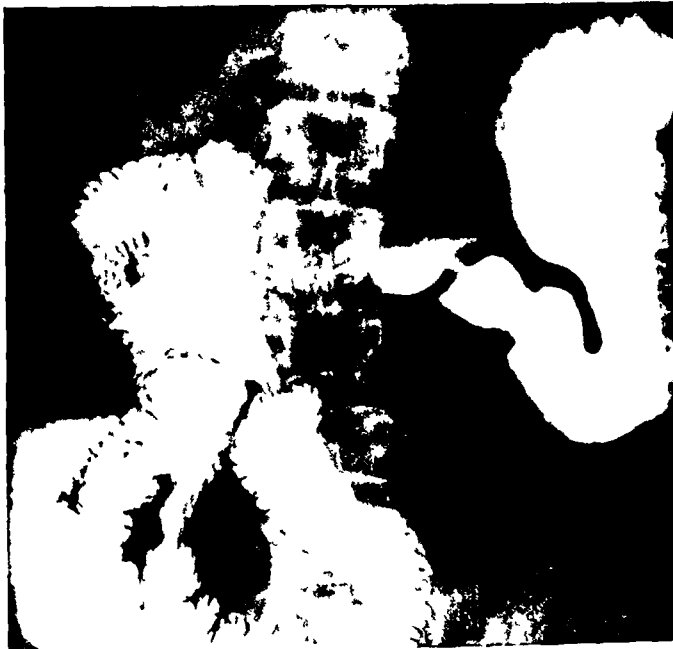


FIG 383 Anomaly of the duodenal curve The second portion of the duodenum is directed to the right where it joins the jejunum on the right side of the abdomen

of the duodenum. The evidence was interpreted as indicating pressure by a mass extrinsic to the duodenum. As noted at operation there were massive adhesions in the pyloroduodenal region and the gallbladder was found pressing down upon the duodenum.

DUODENAL ATRESIA

Rare types of congenital anomalies of the duodenum have been described, such as atresia, and stenosis from the compression of an annular pancreas surrounding the duodenum.

The first description of atresia of the duodenum was apparently that of Calder¹³ in 1733. According to Cautley, Aubrey in 1803 described a case of congenital duodenal stenosis and Meckel in 1812 quoted two instances of congenital occlusion of the duodenojejunal junction. In Jacobi's¹⁴ case, described in 1877 a child weighing 5½ pounds lived 4 days. At autopsy, the stomach and upper part of the duodenum were dilated. There was occlusion of the lower part of the duodenum and intestine.

Spriggs¹ collected 328 cases of congenital intestinal occlusion to which he added a description bearing on the subject. One case was that of a stenosis of the duodenum by a narrow region about a quarter of an inch long fibrous to the touch. The duodenum above the papilla was greatly dilated. In another case the stenosis occurred at the duodenojejunal flexure and scarcely admitted a fine probe. In other cases the cause was an atresia in the second portion of the duodenum. In one remarkable case of atresia the child survived for 9 months apparently as a result of absorption from the stomach.

The most frequent areas of congenital occlusion, according to Spriggs are (1) in the duodenum near the papilla of Vater, (2) at the duodenojejunal flexure, (3) in the lower ileum and (4) in the rectum (most frequently).

A review of the older literature on congenital atresia of the duodenum is to be found in the article by McDonald,¹⁶ who added a case of his own.



FIG. 384. Angulation of the second portion of the duodenum due to adhesions.

Additional case reports of atresia of the duodenum are to be found in the contributions of Thorndike,¹ Cautley¹⁸ and Kaldor.¹⁹

The Origin of Duodenal Atresia. According to Tandler,⁹ the duodenum is particularly predisposed to the development of congenital atresia. He doubted the significance of fetal peritonitis or axial rotation as a fundamental explanation of the condition. Most of the atresias of the duodenum occur in an area which is least liable to axial rotation, because this region possesses the shortest mesentery. Tandler's explanation based on the embryologic development of the duodenum is as follows: Epithelial occlusion of the duodenum occurs normally in the course of its development. The persistence of such occlusion beyond the physiologic state into later life offers a rational explanation of atresia.

That such atresias are due to errors in development is suggested by the fact that as Bland Sutton stated, they occur most frequently at the "site of important embryological events." Thus, the duodenum

where atresia is most common is the site of a complicated rotation during development. It is also the location of the fetal anlage for the outgrowth of liver and pancreas.

Roentgen Findings The roentgen findings in this condition depend on the occlusion of the duodenum beyond the bulb. The congenital stenosis as a rule produces an entire obliteration of the involved area of the duodenum. As a result, roentgen examination discloses two essential findings:

1. Marked dilatation of the stomach and the duodenum up to the occluded region.

2. As a rule, complete retention of the barium. Obviously, the more distal the region of stenosis, the greater the segment of the duodenum which will be involved in the secondary dilatation.

3. In the roentgenographic examination for suspected atresia of the duodenum a scout film is often sufficient. This will show the enormously gas-distended stomach and duodenum and the absence of gas distal to the obstruction. Such examination should be made without the preliminary administration of an enema, since some gas may be introduced in this manner and perhaps create the impression of only a partial occlusion. If any gas is present distal to the dilated stomach and duodenum, it is possible that the obstruction may then be due to a partially perforated diaphragm, an obstructing band or a partial volvulus. Then it may be safe to administer a small amount of barium by mouth. The suspension should be thin so as to prevent any inspissation which may occlude a narrow opening in the duodenum and change a mild obstruction into one that is complete. In some cases it may be desirable to remove most of the barium suspension from the stomach by means of a nasal catheter after completion of the examination, particularly if prompt surgical intervention is contemplated. Also, the barium suspension, if permitted to remain within the stomach for an unnecessarily long period of time, may regurgitate by way of the hypopharynx into the lungs.

Roentgenographic examination in the

erect position in the presence of a congenital occlusion of the duodenum, when a considerable amount of fluid is present in the stomach, may show only a wide fluid level capped by a large air bubble without any gas in the small intestine. However, the sharply delimited area in the duodenum at the site of the atresia may not be demonstrable. Examination in the prone position may succeed in forcing some of the air into the duodenum and thus show the region of demarcation. If this procedure is not entirely successful, then some of the fluid may be suctioned off by way of a nasal catheter. This fluid may represent not only gastric secretion but also perhaps retained water or milk which may have been given to the infant before the obstruction was suspected. Under no circumstances should air be introduced directly into the stomach in order to demonstrate more clearly the sharply defined area of obstruction, because of the added danger of an acute perforation.

Caution in the recognition of a partial diaphragm at the time of operation when it is the cause of congenital duodenal obstruction has been emphasized by Krieg.²¹ The external appearance of the bowel wall in the region of the diaphragm may be normal to inspection and palpation except for some dilatation and hypertrophy, varying, of course, with the degree and the duration of the obstruction. The importance of realizing the fact that there may be an absence of any external evidence of serosal pathology is that otherwise the existence of the diaphragm may not be recognized at the time of operation. The diaphragm was proximal to the ampulla of Vater in 45 per cent of cases, opposite the ampulla in 20 per cent, distal to it in 25 per cent and at the duodenojejunal junction in 10 per cent. It is composed of a continuation of the mucous membrane and the submucosa. A few muscle fibers have been found in the base of the diaphragm, and in Seidl's case (quoted by Krieg) there were islands of pancreatic tissue. Depending on the completeness of the occlusion by the diaphragm or grada-

tions in the size of the perforation within it symptoms of obstruction may develop within a few hours after birth or a number of years later

One of the earliest cases showing the

roentgen appearance produced by duodenal atresia was published by Bolling At 6 hours the duodenum showed huge dilatation and complete retention A duodeno-jejunostomy was done, with a successful



FIG 385 (A *Top*) Congenital atresia of the duodenum (immediately after ingestion of barium) The stomach is on the right side due to a congenital anomaly of position Note that the duodenal bulb is abnormally dilated and that none of the barium has passed beyond it (B *Bottom*) Same patient Total obstruction in the stomach and the duodenum at 24 hours



result Roentgen examination, 6 weeks later, showed excellent functional emptying through the anastomosis. He also included the autopsy specimen of another case, which demonstrated the anatomic nature of the constriction. In this case the cause was a diaphragm apparently in the second portion of the duodenum.

Additional characteristic pictures of the roentgen appearance in congenital atresia of the duodenum are to be found in the case reports by Thompson²¹ and Wangenstein.²¹

Illustrative Case The following is an example of atresia of the duodenum.

A newborn infant, a patient of Dr Stringfield, continued to vomit bile-colored fluid until the time of its death about 7 days after birth.

The autopsy findings were "Abdomen. The stomach was distended. Position is on the right side, the cardiac opening being under the dome of the right diaphragm. The pyloric opening was pointing to the left into the duodenum, which was likewise distended and ended in a saclike pouch at the second portion where there was no opening.

"There was a small fibrous band 1 inch in length connecting the end of the second portion of the duodenum with the beginning of the jejunum, which also started as a closed pouch. The jejunum, ileum and colon were partly filled with blood. The spleen was situated on the right side and consisted of six small, lobulated, round pieces of splenic tissue which have failed to fuse together. Both kidneys were normal as to size and position. The pancreas was normal. The cecum and the ascending colon showed an absence of mesenteric attachment and were freely movable over the entire peritoneal cavity."

Roentgen examination made at the onset of the child's symptoms revealed the following. The film taken immediately after ingestion of barium showed a stomach of increased size. It was transposed in position, being on the right side. The duodenal bulb was visualized and was very large but otherwise exhibited no evidence of deformity (Fig 385 A). The completeness of the obstruction beyond the duodenal bulb is shown by the fact that at examination 24 hours later (Fig 385 B) the stomach and duodenum show a total retention of all the barium, not a trace having escaped distally beyond the duodenal bulb. A complete obstruction of this character

in a newborn child could be explained only on the basis of an atresia of the duodenum beyond the bulb.

ANNULAR PANCREAS AS A CAUSE OF CONSTRICTION OF THE DUODENUM

According to Lecco,²⁵ the first description of "pancreas annulare" was by Becourt in 1830. This was followed by a contribution to the subject by Moyse in 1852. The condition is distinguished from that of the normal pancreas by an annular portion which arises from the dorsal lobe of the pancreas and embraces the duodenum. The arrangement of the ducts in this type of pancreas is similar to that in the nonannular pancreas.

In a series of 105 specimens of the adult human pancreas which Baldwin²⁶ studied at the anatomical laboratory at Cornell University, he found one example of annular pancreas which completely encompassed the duodenum. He reviewed the literature up to the time of his own report in 1910 and found that there had been only 8 similar cases previously reported. The explanation is to be sought in the embryologic origin of the pancreas, arising from the duodenal wall in the form of two anlagen, a dorsal and a ventral one, the latter consisting of two parts, left and right. The left half, which ordinarily atrophies, may persist as a ring of pancreatic tissue encircling the duodenum.

Examples of constriction of the duodenum by an annular pancreas have been described by Summa,²⁷ Wanke,²⁸ Howard,²⁹ Brines,³⁰ Lehman,³¹ Stoffer³² and Conroy and Woelfel.³³

ANOMALIES OF INTESTINAL ROTATION AND DUODENAL OBSTRUCTION

Congenital anomalies of intestinal rotation may predispose the small bowel to volvulus, thus giving rise to the clinical picture and roentgen evidence of duodenal obstruction. The cause of the intestinal obstruction may be produced either by



FIG 386 Marked dilatation of the first and the second portions of the duodenum secondary to obstruction produced by congenital bands as well as by a diaphragm containing a small 2 mm opening in the middle (A, *Top left*) Appearance immediately after ingestion of the barium (B, *Top right*) Appearance 1 hour later (C, *Bottom, left*) Appearance 3 hours later (D, *Bottom right*) Appearance at 24 hours The tremendous distention of the proximal portion of the duodenum is still present Even at this stage only a small amount of barium has escaped beyond the distended duodenum

volvulus of most or all of the intestine which arises from the midloop or in the persistence of peritoneal bands which constrict the duodenal channel or in a combination of both these abnormalities^{34 35}

The underlying cause of the obstruction may be suspected on the basis of the following roentgen features

1 If the obstruction is incomplete a barium suspension administered by mouth

may enable one to demonstrate that the proximal loops of the jejunum are abnormally placed on the right side of the abdomen

2 At 24 hours the barium-filled cecum and ascending colon may be present on the left side, because of an associated congenital anomaly in the position of the colon

3 In the event that the duodenal obstruction is complete and there is serious contraindication to the ingestion of barium, then the colon may be visualized by means of a barium enema. The visualization of a congenital abnormality in the position of the colon and particularly of the cecum may lead one to suspect that the duodenal obstruction is due to an anomalous position of the small intestine, resulting in kinking or actual volvulus at its junction with the duodenum

Obstruction of the duodenum in an infant apparently due to congenital bands associated with a perforated diaphragm, is shown in the following case

P G, female, 2 years old, was a premature baby born at Bellevue Hospital where she remained until she was $2\frac{1}{2}$ months old. During this time she gained weight very satisfactorily, but the nurses recalled that she had to be propped up after each feeding because of her tendency to vomit. From the time of her discharge she had a number of episodes of vomiting which were controlled medically in a few days. Physical examination of the abdomen was essentially negative.

Roentgenographic examination revealed enormous dilatation of the proximal portion of the duodenum, due to an obstructive lesion beyond it. Figure 386 A showed the appearance promptly after the ingestion of barium. While the stomach was of somewhat increased size, it was primarily the dilated duodenum that was of clinical significance. At the end of 1 hour (Fig 386 B) a constricted portion of the duodenum could be seen distal to the markedly distended proximal segment. Very little barium had escaped into the distal loops of the small intestine. At the end of 3 hours (Fig 386 C) the proximal portion of the duodenum was even more dilated than in the preceding observation. Only a small amount of barium had passed beyond the confines of the dilated duodenum. The marked distention of

the proximal portion of the duodenum and the degree of obstruction beyond it was particularly apparent in the observation at 24 hours (Fig 386 D).

The preoperative roentgen diagnosis was marked dilatation of the proximal portion of the duodenum secondary to an obstructive lesion distal to it. It was quite obvious that the obstruction was incomplete. Also, it had been noted during roentgenographic examination of the colon by means of a barium enema that the cecum was in the right lower quadrant. Also, in the 24-hour observation (Fig 386 D) the cecum occupied the right lower quadrant. Because of the normal position of the cecum I believed that constricting bands as a cause of the duodenal obstruction were less likely than a partial atresia, perhaps due to a perforated diaphragm.

At operation the first and the second portions of the duodenum were found to be markedly dilated to about the diameter of the stomach. The length of the dilated segment was approximately from 8 to 10 cm. Upon following the duodenum to the point of obstruction, it was found that there were numerous adhesive bands which caused the duodenum to double up on itself. There was no ligament of Treitz, and the duodenojejunal junction occurred close to the right side of the abdominal wall. Some of the adhesive bands involved the region of the hepatic flexure of the colon. A second point of obstruction by a band involved the middle portion of the ileum. No other lesions were found in a complete exploration of the gastro-intestinal tract. After the bloodless bands which obstructed the duodenum were freed, the entire small intestine could be shifted to the left portion of the peritoneal cavity. The stringlike adhesion which constricted the ileum was incised without difficulty.

The child made an uneventful recovery and appeared to be doing fairly well clinically. Nevertheless, it was significant that roentgenographic examination one month later showed an identical degree of obstruction.

Several months later, persistence of the obstruction necessitated further surgical intervention. "The duodenum was tremendously dilated, thickened, and strong peristaltic waves were visible. The duodenum was opened. The third part of the duodenum was closed by a thick diaphragm. A small 2-mm.-sized opening was present in the middle of the diaphragm." This perforation in the diaphragm had permitted the escape of gastric content into the distal small intestine.

REFERENCES

- 1 Dwight, T Notes on the duodenum and the pylorus *J Anat & Physiol* 31:16, 1891
- 2 Harman, N B The duodenojejunal flexure, its variations and their significance, *J Anat & Physiol* 32 665, 1898
- 3 Ochsner, A J Constriction of the duodenum below the entrance of the common duct and its relation to disease, *Ann Surg* 43 80, 1906
- 4 ——— Further observations on the anatomy of the duodenum *Am J M Sc* 132 1 1906
- 5 Boothby W M The so called "Ochsner muscle" of the duodenum *Boston M & S J* 157 80, 1907
- 6 Bloodgood, J C Dilatation of the duodenum in relation to surgery of the stomach and colon, *JAMA* 59 117 1912
- 7 Haley J C and Perry J H Further study of suspensory muscle of duodenum *Am J Surg* 77 590 1949
- 8 Miyake H Das "primäre duodenum" als Ursache von Kolikanfällen ein neues Krankheitsbild *Arch klin Chir* 122 269 1922
- 9 Harris M L Constrictions of the duodenum due to abnormal folds of the anterior mesogastrium *JAMA* 62 1211 1914
- 10 Niles W Congenital fixation of the duodenum by hepatoduodenal membranes (Harris bands) *M Clin North America* 8 19 1924
- 11 Duval P, Roux J C and Beclere H La periduodenite sus meso colique essentielle *Arch mal app digest* 13 564 1923
- 12 Buckstein J The duodenogram a new method of visualizing the entire duodenal contour *JAMA* 84 510 1925
- 13 Calder J Two examples of children born with preternatural conformations of the guts *Med Essays & Obst Soc Edinburgh* 1 203 1733
- 14 Jacobi A Congenital occlusion of the duodenum *Tr New York Path Soc* 2 3 1877
- 15 Spriggs N I Congenital intestinal occlusion an account of twenty four unpublished cases with remarks based thereon and upon the literature of the subject *Guy's Hosp Rep* 66 143 1912
- 16 McDonald A L Congenital atresia of the duodenum, *Am J M Sc* 146 28, 1913
- 17 Thorndike A, Jr Duodenal atresia and stenosis in infancy, an important diagnosis—case reports, *Boston M & S J* 195 763 1927
- 18 Cautley E Duodenal stenosis, *Brit J Child Dis* 16 65, 1919
- 19 Kaldor J Atresia of the duodenum and duodenal diverticula, *Ann Surg* 89 6, 1929
- 20 Tandler, J Zur Entwicklungsgeschichte des menschlichen Duodenum in frühen Embryonalstadien *Morphol Jahrb* 29 187, 1900
- 21 Krieg E G Duodenal diaphragm *Ann Surg* 106 33 1937
- 22 Bolling R W Complete congenital obstruction of the duodenum duodenojejunostomy at nine days *Ann Surg* 83 543 1926
- 23 Thompson C V Congenital atresia of the duodenum with report of a case *California & West Med* 26 487 1927
- 24 Wangenstein O H Elaboration of criteria upon which the early diagnosis of acute intestinal obstruction may be made with special consideration of the value of x-ray evidence *Radiology* 17 44 1931
- 25 Lecco T M Zur Morphologie des Pankreas annulare *Sitzungsberichte der Akad d Wissensch* 119 391, 1910
- 26 Baldwin W M A specimen of annular pancreas *Anat Rec* 4 299 1910
- 27 Summa H Pankreas annulare in report on the pathological exhibit Missouri State Medical Association, *JAMA* 35 43 1900
- 28 Wanke R Duodenalanomalien im Röntgenbild und ihre klinische und therapeutische Bedeutung *Fortschr Geb Röntgenstrahlen* 39 249 1929
- 29 Howard N J Annular pancreas *Surg Gynec & Obst* 50 533 1930
- 30 Brines O A Annular pancreas involved in acute hemorrhagic pancreatitis *Ann Surg* 92 241 1930
- 31 Lehman E P Annular pancreas as a clinical problem *Ann Surg* 115 574 1942
- 32 Stofer B E Annular pancreas a tabulation of the recent literature and report of a case *Am J M Sc* 207 431 1944

- 33 Conroy, C F , and Woelfel, G F An-
nular pancreas A report of two cases,
Surgery 29 902, 1951
- 34 McIntosh, R , and Donovan, E J Dis-
turbances of rotation of the intestinal
tract, clinical picture based on observa-
tions in 20 cases, Am J Dis. Child
57:116, 1939.
- 35 Glover, E D , and Hamann, C A In-
testinal obstruction in the newborn due
to congenital anomalies, Ohio State M J
36 833, 1940

Diverticula of the Duodenum

INCIDENCE AND TYPES

One of the earliest reports of a duodenal diverticulum was that of Morgagni,¹ who described a diverticulum of the duodenum two fingers breadth distal to the pylorus, the orifice of which was large enough for the insertion of a finger. It was not enclosed by any tunic except the serosa. There was no trace whatever of ulceration past or present. He also described diverticula in the goose, and other authors found it in the horse, dog, hog and rodent.

According to Buschi,² Fleischman described 3 cases of duodenal diverticula in 1815. In 1 case, of a man 64 years old, there were two diverticula near the papilla of Vater. In the second case there were four diverticula near the papilla. In the third case, that of a man 28 years old, several small diverticula were also found near the papilla. In addition, Buschi made a resume of all the cases of diverticula of the duodenum in the literature up to the date of the publication of his paper in 1911. To this he added 3 cases of his own.

Meckel described a hernia of the inner membrane and the formation of a rounded tumor (in the intestine) termed a "false diverticulum (d. spurium)." He stated that they were to be found frequently in the duodenum.

Perry and Shaw³ made an exhaustive study of diseases of the duodenum based on an analysis of 17,652 autopsies. They described two varieties of pouches originating in the duodenum. In one type, the wall is either perfectly normal or has been altered

by distention, so that the wall is thinned or the muscle fibers separated. In the second type, the pouch is the result of previous ulceration of the mucosa, or it develops because of adhesions to the serosa. In the first group the mucosa is normal and the serosa free of adhesions. Because of the thinning and the separation of the muscular layer there is a herniation of the mucosa. Diverticula are usually single, only one of their cases showing two such diverticula. They found the most common location to be the papilla of Vater. The age of the youngest patient was 34 and that of the oldest 84 years. The lesions had produced no symptoms and in no case were they the cause of death.

Because of the fact that the site of predilection of duodenal diverticula is near the papilla of Vater, Letulle⁴ described them as "diverticules perivateriens." Their freedom from any adhesions distinguished them from diverticula secondary to inflammation of the duodenum. Letulle considered such diverticula to be of congenital origin, developing from the primitive intestine at the same time as the liver and the pancreas.

The remarkable frequency of duodenal diverticula is indicated by Baldwin,⁵ who out of a series of 105 duodenums examined in the anatomical laboratory of Cornell University Medical College found 15 cases. In addition Baldwin gave a resume of all the cases of duodenal diverticula reported in the literature at the time of his personal contribution in 1911. Exclusive of his own series he found descriptions of only 67 cases. However, Baldwin rightly believed

that this by no means gave a true insight into the frequency of duodenal diverticula since many are either overlooked or not reported. Usually single, as many as five diverticula were present in one duodenum. He found that while they might occur anywhere throughout the duodenum, they were more common in the second portion. They were usually on the mesenteric side, Baldwin having found all his cases to be so situated.

ETIOLOGY

Perry and Shaw considered the cause for the development of these diverticula to be increased intraduodenal pressure. This is brought about by the fact that the pylorus prevents regurgitation of intestinal contents when the duodenum is contracting thus exaggerating the mechanical pressure. In addition, there is a weakness of the muscular wall in the second portion of the duodenum produced by the oblique insertion of the common bile duct. This factor would tend to make such an area more amenable to the effect of the increased intraduodenal pressure.

The congenital theory of the origin of duodenal diverticula is supported by the work of Baldwin and Buschi. Baldwin found in an examination of 100 duodenum that, in 60 cases, there was a distinct hollowing of the duodenal wall at the site of the major papilla, with the common bile and the main pancreatic ducts situated in this depression. Such findings suggest the persistence of the original diverticulum from which both the liver and the ventral anlage of the pancreas originate, and favor the congenital origin of diverticula in this region.

The congenital origin of duodenal diverticula received strong support from Buschi. His main reasons for assuming the congenital nature of the diverticula were

- 1 The duodenum is that portion of the intestine which during fetal life undergoes the most profound changes because of the fact that the ducts of the pancreas and liver

develop from it. It would therefore not be strange for developmental anomalies to arise in this region. In two of his cases he found dilatation of the common bile duct of a congenital nature.

- 2 He quoted a case reported by Shaw in which a duodenal diverticulum was found in a newborn child. This diverticulum was associated with a congenital occlusion of the duodenum.

- 3 The fact that duodenal diverticula may be associated with other congenital diverticula, as in the case reported by Falconer,⁶ in which a diverticulum of both the stomach and the duodenum were found.

- 4 The presence of areas of accessory pancreatic tissue distal to the pylorus.

- 5 The observations of Lewis and Thyng⁷ that during the second month of fetal life in mammals there are numerous epithelial excrescences throughout the entire length of the intestine. These are particularly marked in the duodenum and jejunum.

- 6 The absence of any mechanical cause for the formation of such diverticula, such as ulcers, tumors or adhesions. Thus in the three personal cases which he reported there was no evidence of any of these mechanical factors. There was no evidence of scars or of inflammatory changes in neighboring structures.

As previously stated Morgagni noted diverticula of the duodenum in various forms of lower animal life.

ROENTGEN DIAGNOSIS

Roentgenologically, a diverticulum of the duodenum has the appearance of a rounded sac, as a rule connected by a narrow neck with the duodenum where it originates. Strands of normal mucosa may be seen entering the neck of the diverticulum. Rarely, mucosal folds may be noted within the sac itself. Diverticula of the first portion of the duodenum are extremely rare. Those found in this area have almost invariably been sacular dilatations secondary to an ulcer and prestenotic in location. I have personally never seen a case of a primary diverticulum.

of the duodenal bulb. Practically speaking, diverticula may be found anywhere in the rest of the duodenal curve up to and including the duodenojejunal junction. The earliest cases of a diverticulum of the duodenum with a roentgenologic description are those of Case⁸ and Forssell and Kay.⁹

The site of predilection for the origin of diverticula is the perampullary region. The diverticulum may then be noted extending horizontally inward about midway between the duodenal bulb and the transverse portion of the duodenum. As a rule, the diverticula are single and originate along the mesenteric border of the duodenal curve. In some cases they may be multiple and in one instance Spriggs and Marver¹⁰ found six diverticula of the duodenum as well as others in the jejunum. The diverticulum may at times be freely movable. It may exhibit superficial variation of contour and as a rule is not tender to localized palpation unless inflammatory changes have occurred within it. In rare cases a diverticulum may be noted originating from the lateral border of the duodenal curve. In the erect position, the diverticulum may show a fluid level capped by gas.

Frequently, diverticula are discovered accidentally in routine examination and have no clinical significance. At times however, a diverticulum may give rise to symptoms as the result of ulceration. In cases of upper abdominal pain localized tenderness over the site of a diverticulum in the absence of any other discoverable cause may be suggestive of inflammation. That diverticula are capable of producing serious disturbances is shown in the report by Roenthal,¹¹ who described the findings at autopsy in a 70 year old woman of a diverticulum of the second portion of the duodenum which had compressed the distal end of the common duct, causing dilatation of the duct and because of pressure on the pancreatic duct had caused necrosis of the pancreas. In Bauer's¹ two cases of duodenal diverticula there were definite disturbances which could be traced to their presence.

Thus, in one case, checked by autopsy findings, the diverticulum near the papilla of Vater was inflamed and covered with a viscid layer of mucus which it was difficult to detach. There was inflammation and swelling at the papilla of Vater, which was closed off by a plug of mucus. The bile duct was dilated and the gallbladder distended. Icterus was present. Apparently the diverticulum had been responsible for a duodenitis and inflammatory changes of the common bile duct. Also, in Akerlund's¹³ case of a diverticulum of the ampulla of Vater, operation revealed a necrotic mass in the head of the pancreas close to the duodenal wall at the origin of the diverticulum. The etiologic relationship apparently depended upon the development of a diverticulitis with swelling of the papilla of Vater and secondary stasis and dilatation of the ducts. Lucman¹⁴ also reported a case of perforation of a duodenal diverticulum into the head of the pancreas.

Nicholson¹ described the findings at autopsy showing the compression produced by a diverticulum of the second portion of the duodenum on the distal end of the common duct. It is interesting that clinically the jaundice which had been produced was of an intermittent character, probably due to variations in the degree of distention of the diverticulum.

Ogilvie¹⁶ reported four cases confirmed by autopsy in which a diverticulum in the papilla of Vater had occluded the pancreatic duct and caused necrosis of the pancreas.

Ordinarily the diverticulum is emptied of its barium content at about the same time as the rest of the duodenal curve. Occasionally however a small amount of barium may remain in the diverticulum longer than elsewhere in the duodenum and in rare instances I have seen a retention of the barium within the diverticulum for many days. Such abnormal retention may be due either to a very narrow normal neck or to edema occluding the neck as a result of inflammation. When the diverticulum

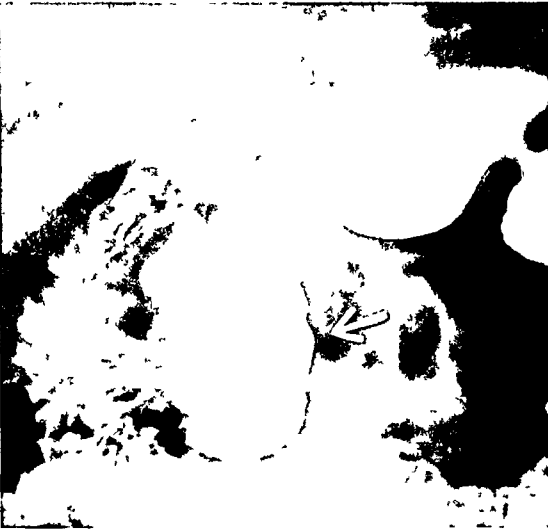


FIG. 387 (*Left*) Diverticulum of the second portion of the duodenum



FIG. 388 (*Right*) Diverticulum of the second portion of the duodenum Note the mucosal structure in the proximal portion of the diverticulum

originates from the lateral wall of the second portion of the duodenum just distal to the bulb, it may simulate the appearance of a gallbladder following a cholecystoduodenostomy. The history, of course, will make differentiation a simple matter. In those cases of spontaneous fistula between gallbladder and duodenum it is the duodenal bulb that is practically always the site of involvement, a region that is peculiarly

immune to the development of diverticula. Moreover, the distortion due to the associated inflammation and the frequent demonstration of the biliary ducts, either due to the presence of air or ingested barium, will all help to make the differentiation readily possible.

Perplexities in differential diagnosis may arise when a diverticulum appears to overlap either the duodenal bulb or the lesser



FIG. 389 (*A, Left, B, Right*) Diverticulum of the second portion of the duodenum Note the variation in the relative position of the diverticulum to the second portion of the duodenum, associated with changes in the peristaltic activity of that segment Note the variations in the shape as well as in the mucosal pattern of the diverticulum

curvature of the stomach. Under such conditions a diverticulum actually originating from the second portion of the duodenum may simulate a niche of the duodenal bulb, and at times, with the film taken in certain degrees of obliquity, may appear as if it were a niche of the lesser curvature of the stomach. It is especially the diverticulum of the duodenojejunal junction, however, that, by overlapping, may closely simulate the niche of a gastric ulcer. Careful fluoroscopic observation and films taken in various degrees of obliquity will lead to a correct differentiation. It is only when a detailed fluoroscopic technic has not been observed and when an attempt is made to reach a diagnosis on the basis of a few films taken without change in the patient's position, that a duodenojejunal diverticulum overlapping the lesser curvature may be falsely interpreted as having been produced by a gastric ulcer. In exceptional cases the duodenogram, as previously described, may aid in the exact localization of a diverticulum by permitting a homogeneous filling of the entire isolated duodenal curve, unobscured by the overlapping stomach.

Differentiation of a diverticulum from an ulcer of the second portion of the duodenum has been previously described, and the characteristic location, the narrow neck of the diverticulum and the occasional changeability of its contour should all aid in its recognition without any difficulty.

Occasionally, a deposit of barium at the inner border of the second portion of the duodenum may be due to retention within a dilated ampulla. In such cases the shadow occupies a characteristic location and as a rule is extremely small amounting to no more than a fleck. Exact differentiation from a very small diverticulum may be impossible, since this region is the site of predilection for their development as well.

Illustrative Cases. The various roentgenologic features of diverticula of the duodenum are demonstrated in the accompanying illustrations.



FIG. 390 Diverticulum of the third portion of the duodenum

Figure 387 shows the most common location of a duodenal diverticulum in the second portion of the duodenum, at its inner border, in the region of the ampulla of Vater. Note the mucosal folds emanating from the duodenum and passing into the diverticulum.

Although the contour of the diverticulum is usually well rounded, it may appear indented along its external border.

Rarely mucosal folds may be noted within the confines of the sac itself (Fig. 388).

A duodenal diverticulum in addition to showing mucosal folds within it may also exhibit minor variations in its relative position primarily the result of peristaltic activity in the loop of duodenum from which it originates. The contour will also vary with the amount of barium within it and also perhaps because of intrinsic peristaltic activity (Fig. 389 A and B). In Figure 389 A the diverticulum originating from the inner border of the second portion of the duodenum is somewhat elongated and forms an angle of about 45° with the loop of duodenum to which it is attached. Mucosal folds are present within it.



FIG 391 Diverticulum of the duodenojejunal junction. With examination in the erect position the diverticulum shows a fluid level capped by gas



FIG 392 Diverticulum of the lateral border of the second portion of the duodenum



FIG 393 Note the multiloculated diverticulum originating from the inferior border of the third portion of the duodenum

In Figure 389 B, with the patient in an identical position the diverticulum is now globular in shape and forms almost a right angle with the second portion of the duodenum. The neck of the diverticulum is also clearly noted. The change in position is apparently secondary to the peristaltic activity in the second portion of the duodenum from which it originates. The mucosal folds within the diverticulum are noted again. These appear to be altered, suggesting that the mucosa of the diverticulum may also be capable of altering its configuration.

Such features in the appearance and the behavior of a duodenal diverticulum offer clear criteria in differentiating it from the niche of an ulcer beyond the bulb.

Less common than a diverticulum of the second portion of the duodenum is one originating from the third portion of the duodenum (Fig 390). Note also the strands of mucosal folds entering the diverticulum.

Less common than either of the two locations already described is the duodenojejunal junction. A diverticulum in this region is shown in Figure 391. Note that, in the erect position, there is a characteris-

tic fluid level. A diverticulum in the duodenojejunal area may, under certain conditions, simulate the niche of a gastric ulcer if it happens to overlap the lesser curvature of the stomach. Differential diagnosis is simple in that change in the patient's position will cause a separation of the diverticulum from the stomach, thereby showing that the suspicious saccular area is not an intrinsic part of the stomach itself.

Although, as a rule, a diverticulum originates at the inner border of the duodenal curve, it may, in rare instances, be noted extending from the lateral border (Fig 392).

A diverticulum may also originate from the inferior border of the third or transverse portion of the duodenum (A. T. male, aged 61). The patient was hospitalized because of an empyema of the right pleural space. The diverticulum was an incidental finding of no clinical significance (Fig 393).

Multiple diverticula of the duodenum are not common. In Figure 394 the proximal diverticulum is at the inner border of the

second portion of the duodenum and the other originates from the third portion. Figure 395 shows three diverticula originating from the third portion of the duodenum.

In the following case the presence of three diverticula of the duodenum was confirmed by autopsy.

H. M., female, aged 65. One year prior to her admission to the hospital, the patient had had an omentopexy and cholecystostomy. The findings at this time were Laennec's cirrhosis and ascites. She became gradually weaker and finally bedridden. Several months before admission, she developed a moderate degree of dysphagia, hoarseness, unproductive hacking cough, vague lower abdominal pains and tingling of the extremities. The dysphagia finally became very marked and the patient was unable to swallow even liquids. She had lost considerable weight. Physical examination of the abdomen was essentially negative except for an upper abdominal scar.

The autopsy findings were as follows. The esophagus is of normal caliber. The mucosa of the stomach is normal throughout. The stomach wall is of normal thickness. A small portion of the stomach near the cardia can be

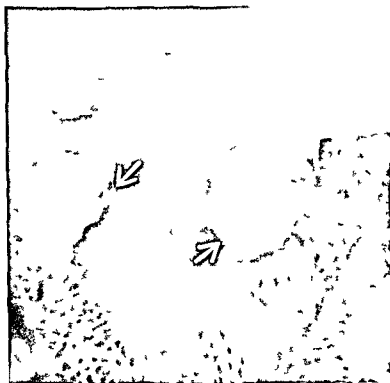


FIG. 394. Two diverticula of the duodenum.

pressed through the esophageal hernial orifice. The duodenum contains three bottle-shaped diverticuli. One measures $2\frac{1}{2}$ cm in length and 1 cm at the neck arising from the medial aspect of the proximal portion of the second part of the duodenum and extending medially anterior to the head of the pancreas and is composed of mucosa grossly. A second diverticulum is found arising just behind the ampulla of Vater. The diverticulum runs behind the common bile duct and in front of the pancreas. This diverticulum is approximately the same size as the first. Its wall is grossly composed of mucous membrane, with a few reddish strands of tissue coursing longitudinally over the mucous membrane. A third diverticulum, measuring 1 cm in depth and $\frac{1}{2}$ cm in diameter at the neck, is found in the midportion of the duodenum, arising from its posterior superior wall, and runs superiorly. It is composed of mucous membrane, grossly

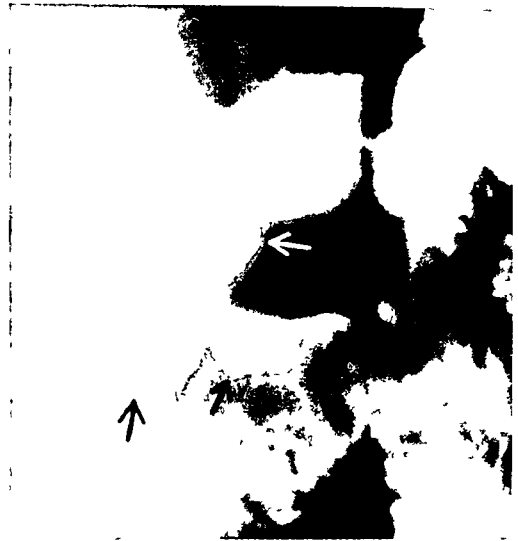


FIG 396 Three diverticula of the duodenum (verified at autopsy)



FIG 395 Three diverticula originating from the superior border of the transverse portion of the duodenum.

The remainder of the small intestine is normal and a moderate amount of white, semisolid, pasty material is found in the lower portion of the ileum. A small amount of fecal material mixed with white, pasty material is present in the ascending and transverse colon. The sigmoid is normal. A few small dilated inferior hemorrhoidal vessels appear through the rectal mucosa.

Roentgen examination (Fig 396) revealed three diverticula at the inner border of the

duodenal curve. The first diverticulum originated at the inner border of the second portion of the duodenum just distal to the bulb. The second diverticulum originated in close proximity to the first in the region of the papilla of Vater. The third diverticulum originated at the superior border of the third, or transverse, portion of the duodenum and was directed upward and slightly inward. As noted above, there was autopsy confirmation of the presence of these three diverticula.

REFERENCES

- 1 Morgagni, J. B. The seat and cause of diseases investigated by anatomy, Trans from the Latin by Benjamin Alexander, London, 1769.
- 2 Buschi, G. Beitrag zur Untersuchung der Duodenaldivertikel Virchows Arch path Anat 206 121 1911.
- 3 Perry, E. C., and Shaw, L. E. On diseases of the duodenum, Guy's Hosp Rep 50 171, 1893.
- 4 Letulle, M. Malformations duodénales diverticules périvateriens, Presse med 7 13, 1899.
- 5 Baldwin, W. M. Duodenal diverticula in man, Anat Rec 5 121, 1911.
- 6 Falconer, A. W. A case of congenital diverticulum of the stomach and duodenum in a physiological hourglass stomach, Lancet 1 1296, 1907.
- 7 Lewis, F. T. and Thyng, F. W. The regular occurrence of intestinal diverticula in embryos of the pig, rabbit, and man, Am J Anat 7 505 1908.
- 8 Case, J. T. Roentgen observations on the duodenum with special reference to lesions beyond the first portion. Am J Roentgenol 3 314 1916.
- 9 Forssell, G. and Kay, E. Ein Divertikel an der Pars descendens duodeni mittels Röntgenuntersuchung diagnostiziert und operativ entfernt Fortschr Geb Röntgenstrahlen 24 48, 1916 1917.
- 10 Spriggs, E., and Marver, O. A. Intestinal diverticula, Quart J Med 19 1, 1925.
- 11 Rosenthal, Theodore. Können Duodenaldivertikel eine klinische Bedeutung erlangen?, Med Klin 4 1421, 1908.
- 12 Bauer, T. Über das Duodenaldivertikel. Wien klin Wchnschr 25 879, 1912.
- 13 Akerlund, A. Duodenaldivertikel und gleichzeitige Erweiterung des Vaterschen Divertikels bei einem Fall von Pancreatitis, Fortschr Geb Röntgenstrahlen 25 540, 1918.
- 14 Lucinian, J. H. Diverticulum of the duodenum perforated into the pancreas: report of a case, Am J Roentgenol 24 684 1930.
- 15 Nicholson, W. M. Jaundice produced by a diverticulum of the duodenum. Bull Johns Hopkins Hosp 56 305, 1935.
- 16 Ogilvie, R. F. Duodenal diverticula and their complications with particular reference to acute pancreatic necrosis, Brit J Surg 28 362 1941.

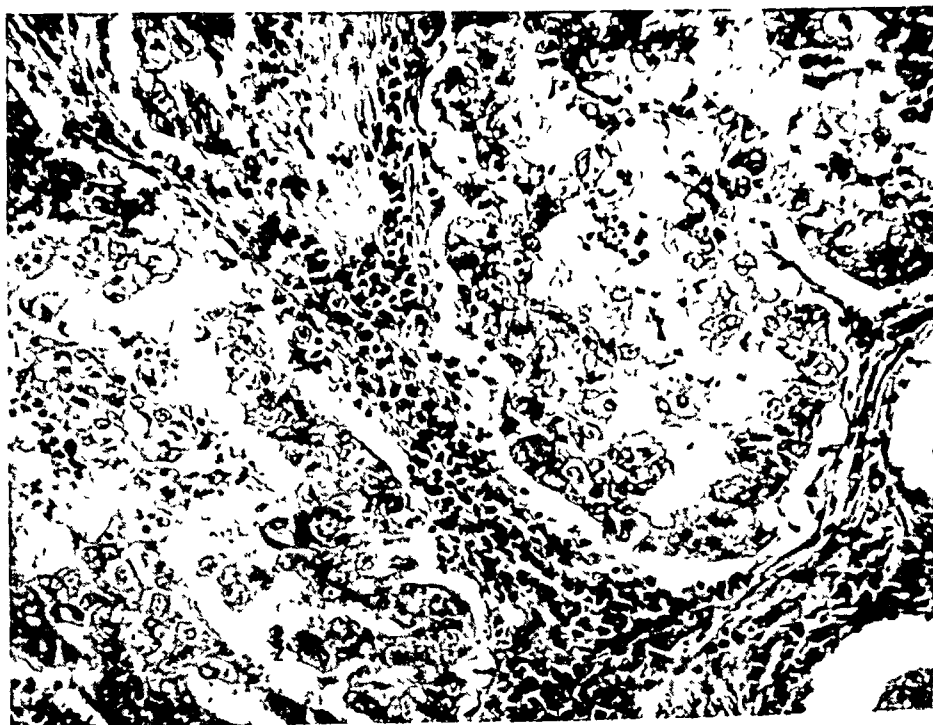
trooduodenal retention at 6 hours. Beyond the zone of obstruction only a trickle of barium may be noted making its escape. However, such stenosis may show a variation on repeated examinations with an interval of weeks in between, that is to



FIG 397 (A, *Top*) Adenocarcinoma in the second portion of the duodenum. Note the translucent area within the second portion of the duodenum produced by the tumor. (B, *Bottom*) Microscopic appearance of the tumor.

say, the original examination may show huge dilatation of the proximal duodenum, with almost complete retention at 6 hours, and yet a later examination may fail to show any marked degree of obstruction. This may be explained by assuming that a partial sloughing of the tumor has occurred in the meantime. When the duodenojejunal area is involved and this region is not clearly visualized because of the overlapping of the barium-filled stomach, one may be led to assume that the original observation did not justify a diagnosis of an organic obstructive lesion as the cause. Although obstruction is a common finding with carcinoma of the duodenum, there may, in some cases, be a surprising lack of such evidence even when the tumor is quite large. This is apt to occur with the bulky polypoid type of tumor because it permits sufficient canalization for the ready escape of the barium.

4 As is the case with polypoid malignant lesions elsewhere, these lesions produce irregularly outlined translucent areas, due to



the protrusion of the growth within the lumen, causing concomitant displacement of the barium

Examples of the irregularity of contour and obstruction produced by intrinsic new growth of the duodenum have been described by Soper¹¹ Mateer and Hartman,¹ Swenson and Levin,¹³ Lichapele and Dumon¹⁴ and Claiborn and Dobbs.¹

Illustrative Cases The following is a case of carcinoma of the duodenum just distal to the bulb

C M, male aged 48 This patient gave a 6 month history of increasing weakness and a loss of about 14 pounds in weight There was no pain at any time except on one occasion when he complained of pain in the back About 1 month before his admission to the hospital he had had a severe hemorrhage in the stool, followed by tarry stools lasting for about 2

days Three blood transfusions were given Physical examination was essentially negative

At operation a robin's egg sized intraduodenal mass was extirpated It was attached to the posterior duodenal wall in the region of the ampulla of Vater but did not occlude the papilla

The pathologic examination was adenocarcinoma of the duodenum A lymph node showed metastatic carcinoma

Roentgen examination (Fig 397 A) showed a rounded intraluminal translucent area within the confines of the second portion of the duodenum A diagnosis of tumor of the duodenum was made Because of its roentgen characteristics I thought that the tumor might be benign As noted in the pathologic study the lesion was an adenocarcinoma Figure 397 B shows the microscopic appearance of the tumor

A tumor of the papilla of Vater may show



FIG 398 Carcinoma of the papilla of Vater Note the irregularly rounded translucent area within the second portion of the duodenum in the anatomic position of the papilla



FIG 401 (A, *Left*) Carcinoma of the papilla of Vater. Note the deformity at the inner border of the second and the third portions of the duodenum and the irregularly outlined translucent areas present mainly in the third portion. (B, *Right*) Same patient. Appearance of the tumor in cross section.

duct to its entrance into the duodenum, it is found to be completely constricted by the mass in the duodenum.

"The pancreas is of average size, pinkish in color, fairly firm in consistency and shows no deformity. The pancreatic duct is found to be somewhat dilated and is traced easily to the head of the organ, where it is found to enter a mass in the duodenum."

The final diagnosis was ampullary adenocarcinoma of the duodenum with metastases to the liver and stenosis of the ampulla of Vater.

Roentgen examination (Fig 401 A) revealed a well-circumscribed, cup-shaped defect at the inner border of the second portion of the duodenum. In addition, there were numerous irregularly rounded translucent areas involving the third portion of the duodenum. The preoperative diagnosis was malignant lesion involving the second and third portions of the duodenum. The possibility was considered that the lesion might represent a tumor of the papilla or an invasion from a carcinoma of the pancreas. As noted at autopsy, the deformity was due to a carcinoma arising from the papilla of Vater. Figure 401 B shows a cross section through the large tumor of the duodenum as well as dilatation of the intrahepatic ducts and a metastatic node in the liver.

A carcinoma originating in the ampulla

of Vater may not only spread distally but also may grow proximally and invade the duodenal bulb.

J. R., male, aged 53. The patient gave an 8-month history of progressive weakness, jaundice and a marked weight loss of from 35 to 40 pounds. The stools were clay-colored. He had no abdominal pain. Physical examination revealed an emaciated, jaundiced, middle-aged white male. The liver was palpable, five fingers' breadth below the costal margin, firm and of irregular outline. The spleen appeared to be moderately enlarged. Chemical studies pointed to an obstructive jaundice. The icteric index was 80.

At operation, approximately 200 cc of clear, yellow fluid was present. The gallbladder was greatly enlarged and measured about 20 cm by 10 cm. The common and cystic ducts were enormously dilated. The liver was of normal size, dark-green in color and firmer than normal. An indurated mass 3 cm by 5 cm was present in the region of the head of the pancreas, in the curve of the second portion of the duodenum. A resection of the head of the pancreas was done, including the entire duodenal curve, with a choledocho-enterostomy and gastro-enterostomy.

The report of the gross examination was "On opening the duodenum, a polypoid mass of tissue extends from the region of the

papilla into the duodenum in all directions for a distance of about 6 cm. The microscopic diagnosis was adenocarcinoma of the ampulla of Vater.

Roentgen examination (Fig 402) shows a smooth defect of the inner border of the entire second portion of the duodenum. The first portion of the duodenum shows a marked deformity with numerous irregularly rounded translucent areas within it. A large area of translucency is present at the junction of the first and second portions of the duodenum. The evidence is that of a tumor involving almost the entire second portion of the duodenum and invading the duodenal bulb.

As noted in the pathologic examination, the tumor was an adenocarcinoma of the papilla of Vater extending in each direction for a distance of about 6 cm.

The difficulty of differentiating between

a primary carcinoma of the second portion of the duodenum and a carcinoma of the pancreas secondarily invading the duodenum is illustrated by the following case.

F B, male, aged 57. The patient gave a 2 month history of intermittent right upper quadrant pain, most marked about one half hour after meals. There was no vomiting or jaundice. He had a severe secondary anemia. Gastric analysis showed an achlorhydria after histamine. On several occasions he had noted tarry stools. He lost 7 pounds in the preceding few months. Physical examination of the abdomen was essentially negative.

Roentgenographic examination (Fig 403) revealed neoplastic involvement of the second portion of the duodenum. Note the deformity of contour of this segment and the rounded area of translucency within it. At operation



FIG 402 Carcinoma of the papilla of Vater. Note the defect at the inner border of the entire second portion of the duodenum as well as the translucent areas within the duodenal bulb as a result of the invasion of this region by the tumor.

'There was a hard nodular mass about the size of a lemon emerging from behind the second portion of the duodenum and extending laterally with digitations into the right lumbar gutter. There were a few hard, pear-sized regional lymph nodes. There were no metastatic deposits within the liver. The remainder of the abdominal organs were of the usual size and in the usual position.'

A small wedge of tissue was removed from the mass at the second portion of the duodenum. A posterior gastro-enterostomy was done.

Pathologic diagnosis of the biopsy specimen: Carcinoma.

Seven months after operation the patient was readmitted with jaundice, dizziness, abdominal pain and itching. Physical examination at this time revealed marked icterus, some emaciation, generalized tenderness of the abdomen and a hemoglobin of 5 Gms. While the evidence was of a carcinoma in-

volving the second portion of the duodenum, the exact origin of the lesion was not determined. The rounded translucent area within the confines of the deformed second portion of the duodenum might suggest that the malignant tumor had originated from the papilla of Vater. The tarry stools during the early period of clinical symptoms and the later development of jaundice might substantiate this view. However, the possibility of a carcinoma of the head of the pancreas breaking through into the lumen of the second portion of the duodenum cannot be eliminated with assurance. In some cases the exact origin of the malignant tumor cannot be determined at the time of surgical exploration, unless a wide resection including the head of the pancreas and the duodenum has been performed. Inspection of the resected specimen may be essential in order to determine the nature of the lesion.

The roentgen appearance of carcinoma of

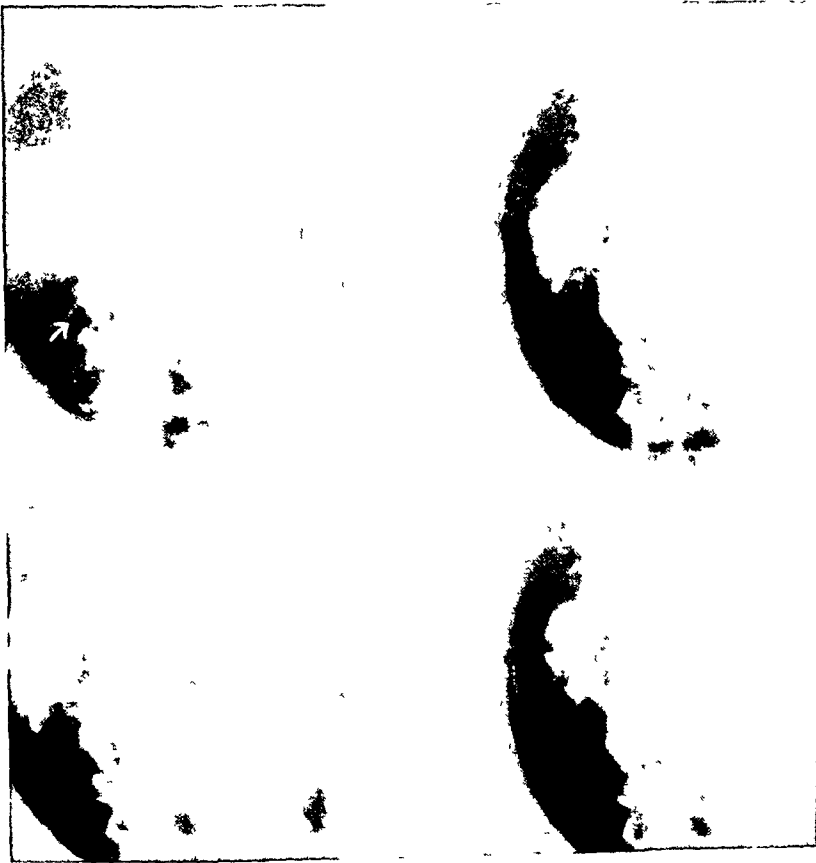


FIG 403 A, B, C, D Carcinoma involving the second portion of the duodenum. Note the marked deformity of the second portion of the duodenum and the rounded intraluminal area of translucency in the different views.

the third portion of the duodenum is illustrated by the next case

H H, male, aged 56 This patient had had an excision of a polyp of the rectum The pathologic report was adenoma Following the operation, however, the patient failed to regain his strength For 3 months he complained of severe pain across the lower abdomen, particularly on lying down Occasionally he also had pain in the back Shortly before operation the pain occurred from 2 to 3 hours after meals and awoke him at night There was no vomiting and no gross blood in the stool He had a severe degree of anemia Physical examination of the abdomen was negative except for a resistance in the epigastrium

Operation revealed a large mass about the size of a grapefruit, with its base in the duodenojejunal fossa The mass was not resectable A biopsy was taken The pathologic report was "carcinoma simplex"

Roentgen examination made prior to operation (Fig 404) revealed an infiltrative lesion involving the distal region of the third or transverse, portion of the duodenum Irregularly outlined translucent areas were present within the zone of involvement The region appeared dilated There was no obstruction to the flow of barium through this region The preoperative roentgen diagnosis

was carcinoma of the third portion of the duodenum

The next case shows roentgen deformities produced by a carcinoma of the third portion of the duodenum

W F male aged 43 Seventeen days and 10 days before admission to the hospital the patient had an episode of weakness collapse with loss of consciousness lasting for a few minutes followed each time by a black stool Two days after the second episode he vomited and from then on continued to do so after every meal He lost from 30 to 40 pounds in the preceding year Stool examination was positive for blood on three occasions Physical examination was essentially negative While on the ward he developed some generalized abdominal pain

Roentgenographic examination (Fig 405 A and B) revealed a deformity of the third portion of the duodenum characterized by irregularity of contour and numerous small rounded translucent areas which I interpreted as being due to a malignant lesion

At operation "A carcinoma of the terminal portion of the duodenum was noted just proximal to the ligament of Treitz This extended distally into the first portion of the jejunum at which point the bowel was ob

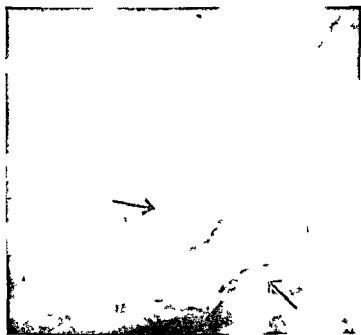


FIG 404 Carcinoma of the third portion of the duodenum Note the irregularity of contour and the intraluminal translucent areas produced by the tumor

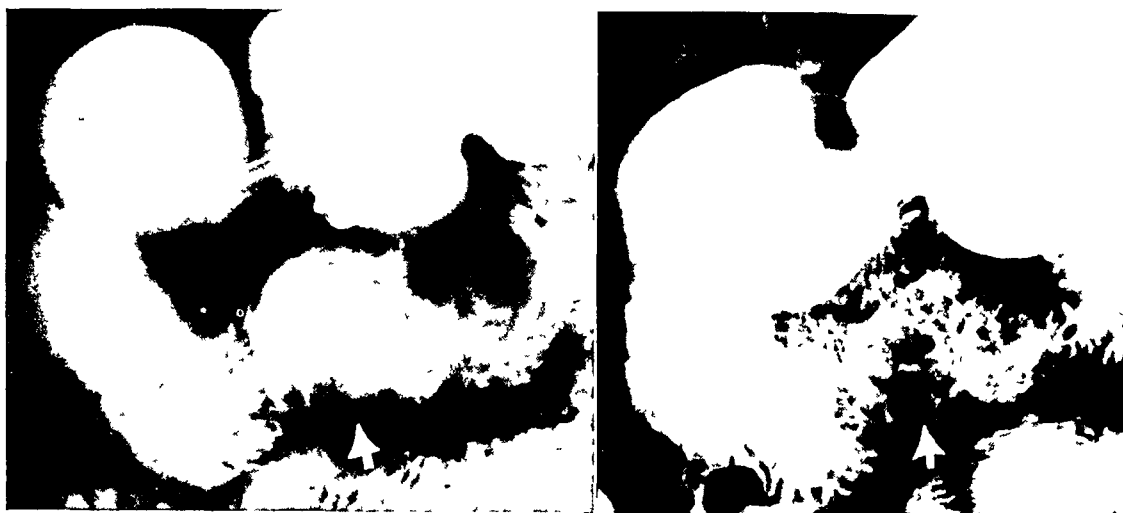


FIG 405 Adenocarcinoma of the third portion of the duodenum (A, *Left*) Note the abrupt change in the mucosal pattern of the third portion of the duodenum This segment is characterized by irregularity of contour but particularly by the many small rounded translucent areas within it (B, *Right*) The abnormal mucosal "relief" pattern of the involved segment is well shown at this time

structed and twisted, and a loop of gangrenous jejunum was noted There were metastases in the regional lymph nodes, numerous metastases in the liver and numerous omental metastases, as well as some in the parietal peritoneum "

Pathologic examination of a biopsy specimen revealed adenocarcinoma

A somewhat similar case of a carcinoma of the third portion of the duodenum is that of A Y, female, aged 65

The patient felt well until one year before admission to the hospital, when she complained of anorexia and gaseous eructations Six months later she noted periumbilical pain made worse after eating and radiating to the epigastrium and the right upper quadrant She vomited infrequently She lost 74 pounds in the preceding year The stools were positive for blood There was a moderate secondary anemia Physical examination of the abdomen was essentially negative

Roentgenographic examination (Fig 406 A)



FIG 406 Adenocarcinoma of the third portion of the duodenum (A, *Left*) Note the deformity of contour and the irregularly outlined areas of translucency within this segment (B, *Right*) Appearance of the resected specimen

revealed no evidence of any organic pathology of the stomach or the duodenal bulb. Examination of the third portion of the duodenum showed definite evidence of deformity of contour and irregularly outlined areas of translucency within this segment. Diagnosis: Malignant lesion of the third portion of the duodenum.

The patient was explored, and the operative report was that no organic lesion was found. Because of the definite roentgen evidence, however, which was corroborated on reexamination of the patient after recovery from the first exploration, she was operated on a second time. The report at operation was as follows: "In the third portion of the duodenum is a large mass, firm and adherent to the pancreas and to the transverse mesocolon below. This involved almost the entire length of the third portion of the duodenum, and many nodes were found in the region of the pancreas and along the superior mesenteric vessels." The lesion was resected (Fig 406 B).

Pathologic diagnosis: Adenocarcinoma of the duodenum.

Note how readily the irregularly outlined translucent areas within the third portion of the duodenum in the roentgenogram may be explained on the basis of the polypoid excrescences of the malignant tumor, which had displaced the barium.

Some of the roentgen features of carcinoma of the third portion of the duodenum are also illustrated in the following case.

J. C. male, aged 38. The patient was in good health until about 2 years before admission to Bellevue Hospital. He complained of abdominal pain made worse by the consumption of fried foods and alcoholic beverages. Fourteen months later he developed recurrent episodes of diarrhea which finally became constant. The bowel movements occurred most frequently during the evening and the night. During the day he had a sensation of rumbling in the abdomen, but there were no bowel movements. The stools were light tan or yellow in color, foamy and with a foul odor. There was no blood or mucus in the stool. He lost 15 pounds during the preceding 2 months.

Roentgenographic examination (Fig 407) revealed some abnormality in the mucosal pattern of the first and the second portions of the duodenum. The bulk of the distortion, however, was in the third portion of the duodenum. The contour was narrowed, ir-

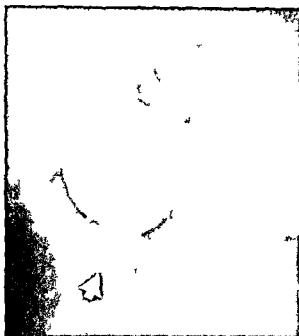


FIG 407 Carcinoma of the third portion of the duodenum, operatively confirmed. Note in particular the narrowing and the irregularity of the configuration of the involved segment.

regular and associated with polypoid changes in this segment. The diagnosis was neoplastic invasion, primarily of the third part of the duodenum. The infiltration of the third portion of the duodenum was verified at exploratory laparotomy. A biopsy was taken from this segment.

Pathologic diagnosis: Carcinoma of the duodenum.

The patient developed a psychosis which required observation on the psychiatric division. He later developed increasing jaundice. Firm, irregular, nontender masses became palpable in the region of the liver, apparently the result of metastatic spread of the primary lesion.

The next case is one of a malignant tumor at the duodenojejunal junction, which produced actual obstruction.

A. F. female, aged 73. This patient had complained of attacks of vomiting for a period of 6 months. The vomitus at first brownish would finally turn green. The attacks of vomiting finally became more frequent and were accompanied by severe pain. Physical examination of the abdomen was essentially negative.

Operation revealed a carcinoma of the duo-

denojejunal junction adherent to the transverse mesocolon, with practically complete obstruction of the lumen of the gut. A resection of the jejunum was carried out. Figure 408 A shows the gross appearance of the tumor.

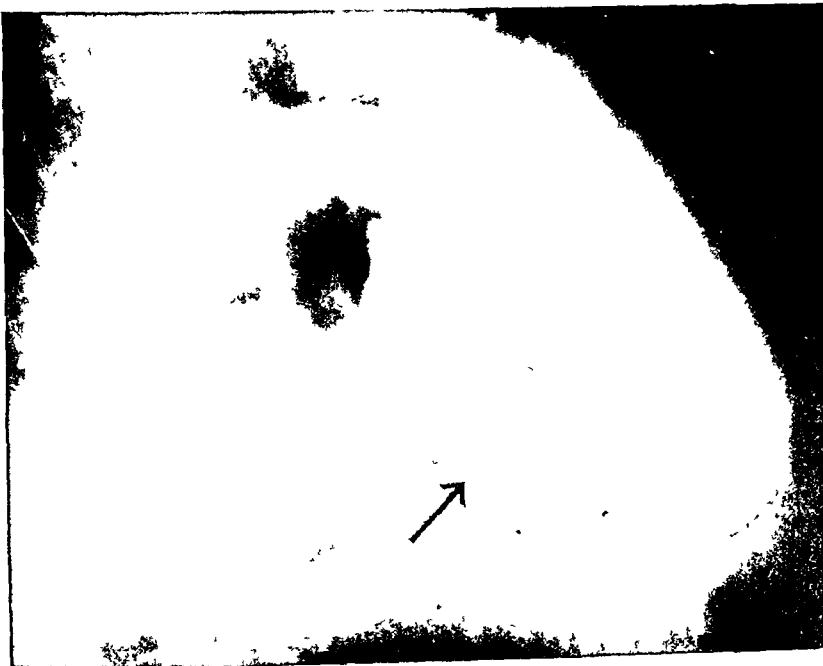
Microscopic examination revealed the tumor to be a metastatic cylindrical-cell adenocarcinoma. As an autopsy was not per-

mitted, the primary focus could not be determined.

Roentgen examination (Fig. 408 B) revealed no evidence of any organic lesion of the stomach or the duodenal bulb. There was a marked dilatation of the entire duodenal curve up to the duodenojejunal junction. Beyond this point, only a trace of barium was to be noted passing through into the jejunum.



FIG. 408 (A, *Top*) Appearance of a malignant tumor of the duodenojejunal junction. Compare with B (*Bottom*). Note the marked dilatation of the entire duodenal curve due to obstruction produced by the tumor at the duodenojejunal junction.



At 6 hours only a small amount of barium was noted beyond the duodenojejunal junction, the rest of the barium being present in the stomach and the dilated duodenal curve.

The roentgen diagnosis was obstructive lesion at the duodenojejunal junction seriously interfering with the emptying of the duodenal curve probably due to a new growth. As noted above, a carcinoma of the duodenojejunal junction proved to be the cause of these roentgen manifestations.

SARCOMA OF THE DUODENUM

Sarcoma of the duodenum is very rare, and of the e leiomyosarcoma is the rarest of all. Prev. Foster and Dennis¹¹ found only 61 authentic cases of primary sarcoma of the duodenum in a survey of the literature up to the time of the publication of their paper in 1935, in which they included a detailed description of a case of their own, confirmed at autopsy.

The first record of a case of a leiomyosarcoma is to be found in the report by von Salis.¹ The case was that of a man aged 40 in whom a clinical diagnosis of pancreatic abscess was made. Autopsy revealed a lobulated tumor in the distal portion of the duodenum and microscopic examination revealed it to be a myosarcoma.

The second well documented case is the one reported by Andersen and Doob.¹² In 1933. In their case there had been a 5 year history of repeated hemorrhages into the intestinal tract and at autopsy a histologically confirmed myosarcoma containing two ulcerations was found, originating from the second and the third portions of the duodenum.

Additional reports were those of Silverstone,¹³ Seymour and Gould,¹⁴ Foshee and McBride,¹⁵ Mayo, Henning and Garland,¹⁶ McCullough,¹⁷ and Shackelford, Fisher and Firor.

The sarcoma by virtue of its origin in the submucosa or muscularis, grows longitudinally by infiltration of the intestinal wall, transforming the involved area into a rigid tube without serious encroachment upon the lumen in the early stages. Ulti-

mately, obstruction is produced as the tumor greatly increases in size. Because of its submucosal localization in the early stage, it is not so apt to ulcerate as is the carcinoma. With increase in the size of the lesion, however, the mucosa is involved and ulcerations develop. Because of the intramural character of the growth, there may be no radiologic evidence of abnormality, at least in the early stages. Thus, in the sarcoma reported by von Salis¹⁷ roentgen examination had failed to establish evidence of abnormality of the duodenum and the reason for this failure was evident from gross inspection of the specimen. The lesion had produced neither narrowing nor dilatation of the lumen and the mucosa was essentially intact. Similarly, in Silverstone's¹³ case, roentgen examination had disclosed no abnormality of the duodenum. At operation, a partly intramural tumor was resected from the third portion of the duodenum and microscopic examination established the diagnosis of myosarcoma. Photographic reproductions of the gross specimen and of the histologic appearance accompany the article.

In some cases, however, a sarcoma may appear radiologically as a polypoid mass (Seymour and Gould,¹⁴ Henning and Garland,¹⁶ and McCullough,¹⁷). A rounded translucent area within the lumen of the duodenum, having the roentgen characteristics of a benign tumor was present in one of the cases reported by Pohlandt.¹⁸ In addition to the translucent area there was a small dense deposit of barium. Examination of the removed specimen showed it to be a metastatic sarcoma the surface of which had ulcerated. The dense fleck of barium apparently represented a nodule in the duodenal tumor.

In the case reported by Sielaff,¹⁹ multiple discrete rounded translucent areas in the distal portion of the duodenum and also in the small intestine were found on roentgen examination in a 19 year old patient shortly after the excision of a nevus on the back. The excised specimen revealed that these



FIG 409 Sarcoma of the second portion of the duodenum. There is nothing in the nature of the deformity that would help to differentiate it from that of a carcinoma.

translucent areas were produced by metastatic melanomas. One of these tumors was associated with a jejunal intussusception.

Illustrative Cases The following is an example of sarcoma of the duodenum.

M. G., female, aged 55. The patient stated that 35 years previously, she had had an at-

tack of jaundice and, from then on, had had attacks of upper-abdominal pain, two or three times a year, finally becoming so severe as to require hypodermics of morphine. The pain became more localized to the left shoulder and left upper quadrant. She vomited frequently and had lost 25 pounds in a short period. With her last attack, the temperature rose to 103°.

Physical examination revealed tenderness and a mass in the right upper quadrant.

At operation, the gallbladder was full of stones. In addition, there was a tumor of the second portion of the duodenum. A biopsy was taken from the tumor mass. The pathologic report was "Examination shows an invasion of the wall by a tumor composed of large, round cells, sarcomatous in nature. The growth, extending to the mucosa in one place, has reached the surface of the lumen and is ulcerated. The nuclei are round or oval, mostly pale, but in some places they present considerable variation in size, shape and staining. Bands of smooth muscle fibers are seen in some parts of the tumor, which may be derived from the muscle coats split up by the tumor. Diagnosis: Large, round-cell sarcoma of the duodenum."

Roentgen examination (Fig. 409) revealed evidence of invasion of the second portion of the duodenum by a tumor.

A lymphosarcoma of the duodenum masquerading as a polyp is illustrated by the next case.

P. S., male, aged 73. The patient entered the hospital complaining mainly of cough and



FIG 410 (A, Left) Lymphosarcoma of the duodenum masquerading as a polyp. Note the intraluminal

translucent area and the spreading of the mucosal folds in the third portion of the duodenum produced by the tumor. (B, Right) Same patient. Note the appearance of the polyp.

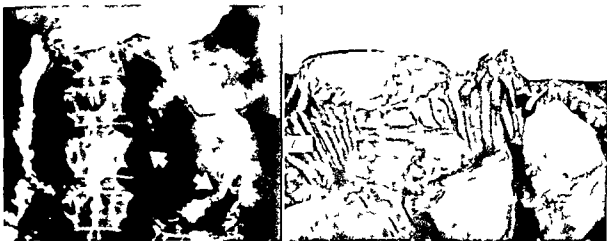


FIG 411 Lymphosarcoma involving the third portion of the duodenum and a segment of jejunum (A, *Left*) Note the marked deformity particularly of the transverse portion of the duodenum. In addition there is an isolated segment of jejunum, showing constriction and distortion of the mucosal pattern (B, *Right*) Appearance of the resected lesion of the jejunum. Note the marked narrowing of the lumen produced by the tumor.

anorexia of 3 months' duration. He brought up mucopurulent sputum, which later became slightly blood streaked. He had lost 27 pounds in the preceding year. Physical examination revealed a hard, 2.5 cm, right supraclavicular nodule, a hard nodule in the right axilla and a few left posterior cervical nodes. Posteriorly at the very base of the lungs, a few medium, moist and coarse rales were heard. The right lobe of the liver could be felt 5 inches below the costal margin. The spleen was definitely palpable.

The autopsy report stated that mediastinal lymphadenopathy extended from the hilar region of the lungs to well above the arch of the aorta. Exploration of the bronchial tree revealed no evidence of tumor. The liver showed a few small, nodular infiltrations. Section of the spleen presented a mottled appearance caused by yellowish raised areas. The pancreas on section showed several small areas of infiltration. On opening the duodenum, a plum sized tumor extending from a long narrow pedicle 1 cm above the papilla of Vater could be seen. On its surface four ulcerations each about $\frac{1}{2}$ to 1 cm in diameter were noted. On section the tumor was similar in appearance and consistency to the lymph nodes. The pedicle of the tumor was not involved. The remainder of the gastrointestinal tract was negative. The histologic diagnosis was widespread lymphosarcoma which also involved the duodenal polyp.

Roentgen examination of the duodenum (Fig 410 A) showed a rounded, intraluminal translucent area of the third portion of the duodenum, with considerable widening in this area and a spreading of the folds. The diagnosis was tumor of the duodenum. As noted at autopsy examination of the polyp showed lymphosarcoma. Figure 410 B shows the gross appearance of the polyp.

A lymphosarcoma of the third portion of the duodenum with a similar lesion in the jejunum was found in the case of N. T. male, aged 52.

The patient was admitted to the medical service with the chief complaints of upper abdominal pain, anorexia, vomiting and a 45 pound weight loss during the preceding 4 months.

Physical examination revealed a hard tender mass in the left half of the epigastric region. There was no hematemesis or melena. During the preceding few days the patient felt chilly and feverish. He ran a febrile course with daily temperature elevations to 102° in spite of streptomycin and penicillin therapy.

Roentgenographic examination (Fig 411 A) was reported as showing an infiltrative lesion involving most of the transverse portion of the duodenum but apparently not involving the duodenojejunal junction. In addition there



FIG 412 Reticulum-cell sarcoma with involvement primarily of the third portion of the duodenum. Note the narrowing and the irregularity of contour of this segment and the destruction of the normal mucosal markings.

is a short segment of jejunum about 5 or 6 inches distal to the ligament of Treitz, exhibiting constriction and abnormality of the mucosal pattern. The evidence is that of a malignant lesion involving the transverse portion of the duodenum. In view of the fact that there is another segment of small bowel apparently representing an invasive lesion, the possibility is suggested that we may be dealing with a lymphosarcoma."

Exploratory laparotomy revealed a massive tumor involving the third portion of the duodenum and the pancreatic region. The tumor was found to consist of numerous nodules of pale white tissue highly suggestive of a lymphomatous type of growth. In addition there was an annular constricting lesion of the jejunum. A resection of the involved segment of jejunum was done. No further operative procedure was feasible.

Pathologic diagnosis of the jejunal tumor was lymphosarcoma, reticulum-cell type.

Figure 411 B shows the gross appearance of the resected mass in the jejunum. Note the constriction of the lumen produced by the mass and the normal mucosal folds to either side. In this case then the diagnosis of lymphosarcoma was made preoperatively because of the fact that two distinct areas were involved by the infiltrative lesion, namely the transverse portion of the duodenum and an isolated loop of the jejunum. As noted above the diagnosis was corroborated by surgical exploration and pathologic examination.

Malignant invasion primarily of the third portion of the duodenum by a reticulum-cell sarcoma is illustrated in the following case.

A C, male, aged 59. Two years before hospitalization the patient developed generalized lymphadenopathy with clinical evidence of mediastinal obstructing nodes. The diagnosis of Hodgkin's disease was made and was substantiated by lymph-node biopsy at another institution. He was treated with nitrogen mustard with quite encouraging results, as indicated by the marked regression in the size of the cervical lymph nodes, relief of dyspnea and increased sense of well-being. During the next 7 to 8 months there was a recurrence of the cervical and the axillary nodes. Again he was treated with nitrogen mustard combined with radiation therapy. He was discharged from the hospital. He returned 2 weeks prior to his last admission, complaining of a dull aching postprandial midepigasttric pain frequently radiating to the back. Vomiting of coffee-grounds material then supervened. The stools were occasionally tarry black in color. He lost 10 pounds in the preceding 2 weeks. On physical examination of the abdomen a tender nodular mass was palpable in the right epigastric area.

Roentgenographic examination revealed the following (Fig 412): "No organic lesion of the stomach or the duodenal bulb. The duodenal curve appears to be widened, and there is evidence of infiltration, particularly of the third or transverse portion of the duodenum. These findings may be explained on the basis of either of the following lesions: (1) a tumor of the head of the pancreas which has secondarily invaded the third portion of the duodenum, (2) a primary tumor of the third portion of the duodenum."

Operative findings: "The stomach and the first portion of the duodenum were of average size. In the retroperitoneal space behind the pancreas and the third portion of the duodenum there was a large tumor mass. The body and the tail of the pancreas could be felt superior to this mass and could be identified as being separate from the mass, however, the head of the pancreas was firmly adherent to it. The curve of the duodenum was widened, and the transverse portion of the duodenum was distended—it lay on top of the retroperitoneal mass, was firmly adherent to it, and the posterior wall of the transverse portion of the duodenum was felt to be involved by the tumor mass. This mass also extended retro-

peritoneally up behind the ascending part of the duodenum and up along and behind the common duct. The gallbladder was tense and distended and on aspiration was found to contain green tinted, thin white bile. The retroperitoneal tumor extended downward as far as the level of the bifurcation of the aorta and the root of the mesentery of the small bowel was pushed toward the right. The tumor was of a bluish purple color and was rubbery in consistency, fairly smooth on the surface, and on section was of a whitish gray color and of fleshy consistency. It bled easily. The tumor was firmly adherent to the aorta. No metastases in the liver.

A gastrojejunostomy was performed, and the gallbladder was anastomosed to the jejunum. A biopsy was taken from the mass.

Pathologic diagnosis: reticulum cell sarcoma.

ROENTGEN DIAGNOSIS OF BENIGN TUMORS OF THE DUODENUM

A benign tumor of the duodenum will show roentgen manifestations essentially similar to those produced by a benign tumor elsewhere in the alimentary tract.

1 The translucent area is, as a rule, of smooth, rounded contour and is entirely within the lumen, similar to that produced by a polyp of the stomach. Moreover, when a benign gastric tumor invaginates through the pyloric ring and appears within the duodenum the roentgen characteristics at that particular time will be exactly those noted with benign tumors arising within the duodenum itself. This rounded translucent appearance produced by a benign tumor of the duodenum was noted in the case of the hemangioma of the duodenum described by Carman⁸ and in the polyp reported by Golden.⁹ Not always, however, is the translucent area produced by the tumor sharply defined and rounded in contour. It may be multilobular in appearance corresponding to the pathologic characteristics of the tumor itself. This occurred in the case reported by Waters.³⁰

2 Because of the lack of infiltration of the wall of the duodenum, its contour remains unaffected and shows no evidence of irregularity or encroachment upon the

lumen. A thin rim of barium may be noted surrounding the tumor itself. This occurred in the case of a fibromyoma in the wall of the duodenum that had not invaded the mucosa as reported by Brdiczka.³¹

3 A moderate degree of mobility may be exhibited by the primary duodenal tumor, so that it may be forced back toward the pylorus by manipulation during fluoroscopic examination.

4 A benign tumor may also produce an extreme degree of obstruction by occluding the lumen. As a result, there may be enormous dilatation of the entire duodenal curve up to the area of involvement, with retention in the stomach as well as in the duodenum at 24 hours and longer. In one of Raiford's³² cases obstruction at the duodenojejunal junction with secondary dilatation of the stomach and entire duodenum, as determined roentgenologically, was produced by a fibroma. An added factor in exaggerating the degree of obstruction produced by a benign tumor may be an associated intussusception.

In addition to deformities of the duodenum produced by malignant and benign tumors there are other rarer causes. Bonomini¹ demonstrated excellent roentgenograms showing the translucent appearance produced by ascariides within the duodenal bulb.

An unusual case of duodenal obstruction was reported by Venables.³³ Roentgen examination with the opaque meal showed marked dilatation of the entire duodenal curve up to the duodenojejunal junction. In spite of vigorous duodenal peristalsis, only a trace of the barium passed into the jejunum. At the end of 3 1/2 hours, however, a considerable amount of barium had reached the jejunum which for a distance of 8 to 10 inches was distended and ended abruptly. The roentgen appearance at 24 hours was identical. Operation revealed a nodular mass infiltrating the wall of the small intestine and completely encircling it where the roentgenogram had indicated obstruction. Examination of a node which was

removed showed the presence of Hodgkin's disease. The tumor itself could not be removed, but this apparently represented a case of Hodgkin's disease primarily in the wall of the small intestine.

The roentgen appearance of a polyp originating within the duodenal bulb is illustrated by the following case.

F F, male, aged 68. One year before admission to the hospital the patient noted bright blood in his stool about once every second or third week. The episodes of rectal bleeding became more frequent. Two and one half months before admission he complained of a postprandial ache in the right upper quadrant. There was no weight loss. Physical examination was essentially negative, except for small hemorrhoids.

Roentgenographic examination when the duodenum was distended with barium (Fig 413 A) disclosed no evidence of a tumor. When partially empty, so that only a thin layer of barium was present, there was a sharply defined rounded translucent area within the confines of the duodenum, apparently hugging the base of the bulb (Fig 413 B). It was my opinion that the cause was a prepyloric polyp intussuscepting through the pyloric ring into the duodenum.

At operation an adenomatous polyp was removed locally from the first portion of the duodenum to which it was attached by a short pedicle. The tumor proved to be an adenomatous polyp of the duodenal bulb (Fig 413 C).

CARCINOID TUMORS OF THE DUODENUM

A carcinoid tumor of the duodenum is quite rare. Lubarsch¹⁵ in 1888 described two unusual tumors of the ileum which he considered to be primary carcinomas. Oberndorfer¹⁶ classified this type of tumor as a carcinoid. He stated that they were usually small, circumscribed, without any tendency to infiltration of the surrounding tissues. He stated further that they grew very slowly, did not metastasize and therefore were apparently benign.

A further advance in the recognition of some of the peculiar characteristics of this type of tumor was made by Masson.¹⁷ In treating sections of two such tumors of the appendix in a bath of ammoniacal silver for 48 hours, the cells became impregnated with the silver and turned black. He was also able to demonstrate identical changes in



FIG 413 Adenomatous polyp of the duodenal bulb (A, *Left*) When the duodenal bulb is filled with barium there is no evidence of the tumor. Examination with a thin layer of barium (B, *Right, top*) reveals a sharply defined rounded translucent area within the confines of the duodenal bulb (C, *Right, bottom*) Gross appearance of the locally excised duodenal polyp.

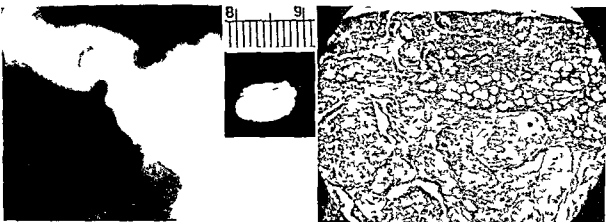


FIG 414 Carcinoid tumor of the duodenal bulb (A, *Left*) Note the sharply defined translucent area within the duodenal bulb produced by the tumor (B, *Center*) Gross appearance of the enucleated tumor (C, *Right*) Microscopic appearance of the carcinoid tumor

some of the cells of the normal intestinal mucosa. Therefore, he assumed that the tumors originated from these "argentaffine cells." For this reason these tumors are sometimes described as argentaffinomas. He wrote: "There is reason to attribute to these cells a special significance which future studies perhaps will allow us to explain."

However, Oberndorfer's view that these tumors always are benign and do not metastasize has not been borne out by experience. Not infrequently they behave like malignant lesions. They occur mainly in the appendix and the ileum. Ariel³⁸ in 1939, in a thorough survey of all of the carcinoids that had been reported since 1930, found 5 cases in which the tumor had originated in the duodenum. Among 84 cases of carcinoid tumors, Porter and Whelan³⁹ found one in the duodenum. Another carcinoid of the duodenum was found at autopsy by Ritchie and Stafford.⁴⁰

All of the 23 carcinoids in the records of the Charity Hospital in New Orleans from 1910 to 1945 inclusive involved the appendix (D. Inganni⁴¹).

Of the 17 carcinoids described by Cruickshank and Cunningham⁴ they found none in the duodenum.

Although they stated in their review of the literature that they had not found descriptions of cases of carcinoid of the duo-

denum, evidently they had overlooked the reports to which I have already referred. Nor was the duodenum involved in the reports of carcinoid tumors by a number of other writers on the subject (D. Albora and Ingegno,⁴² Reid,⁴³ Brown, Bissonnette and Steele,⁴ Grimes and Bell⁴⁶).

Pearson and Fitzgerald⁴ in an analysis of 140 carcinoid tumors from the Mallory Institute of Pathology in Boston found two cases involving the duodenum. No metastases had occurred in either of these cases.

The following is an example of a carcinoid tumor of the duodenal bulb:

V. B. female aged 67. The patient entered the hospital with a history of burning pain in the upper abdomen of 4 months duration. She had had similar episodes during a period of 10 years. She lost 15 pounds during the preceding 3 months. There was no vomiting or tarry stools. Physical examination of the abdomen was essentially negative.

Roentgenographic examination (Fig. 414 A) revealed a sharply defined rounded translucent area within the confines of the duodenal bulb. The preoperative roentgenographic diagnosis was benign tumor of the duodenum. The tumor was believed to be either a polyp or an aberrant pancreatic nodule.

Operation. In the first portion of the duodenum on the anterior wall was a polyp approximately 1 cm. in diameter. It was covered by normal appearing mucosa. There was no evidence of pathology elsewhere in the abdomen. The polyp was shelled out, leaving

the serosa intact. The appearance of the excised tumor is shown in Figure 414 B.

The findings on pathologic examination of the tumor are very interesting.

"Macroscopic examination. Specimen received fresh. It measures 2 x 1.5 cm. The tissue is oval and about 1 cm. in thickness and firm to palpation with equal density throughout. It is well demarcated. The serosal surface has evidence of recent fibrin deposition. The muscular layers are white with no peculiar pattern. The mucosal surface is intact with no signs of ulceration or inflammation.

"Microscopic description. The mucosa is intact but raised by a submucosal mass, consisting of cords and sheets of uniform vacuolated cells with round dark nuclei. There is replacement of Brunner's glands in this area, but no true invasion, and the muscularis is uninvolved. The groups of cells are separated by fine fibrous septa, and cells are not argentophyllic with Fontana's stain.

"Pathologic diagnosis. Carcinoid of the duodenum."

Figure 414 C shows the microscopic appearance of the tumor.

This then is a carcinoid tumor of the duodenal bulb. Clinically and pathologically, it behaved like a benign tumor. Roentgenographically, the deformity was indistinguishable from a polyp.

A benign tumor of the second portion of the duodenum is illustrated by the following case.

R. S., male, aged 31. The patient's only complaints were three episodes of bleeding from the gastro-intestinal tract during the preceding 3 years, the last one occurring in the morning of the day he was admitted to the hospital. The stools were tarry. Physical examination was essentially negative. Roentgenographic examination (Fig. 415 A) revealed an elongated translucent area within the confines of the second portion of the duodenum. A diagnosis of polyp was made.

At operation, the duodenum was opened in the middle of the second portion, and a polyp, about $3\frac{1}{2}$ inches long, was found originating at the junction of the first and the second portions but present within the second portion of the duodenum. A local removal of the polyp was performed. The base was ligated and sutured. The opening in the duodenum, which was longitudinal, was then sutured transversely.

Pathologic diagnosis. Adenomatous polyp of the duodenum.

Figures 415 B and C show the appearance of the polyp. In Figure 415 B, showing the external appearance of the polyp, one may note the ulcer which was the apparent cause



FIG 415 Adenomatous polyp in the second portion of the duodenum (A, Left) Roentgenographic appearance, showing an elongated area of translucency within the confines of the second portion of the duodenum (B, Right, bottom) Gross appearance of the polyp, showing an area of ulceration, apparently the cause of the bleeding (C, Right, top) Cross section of the polyp

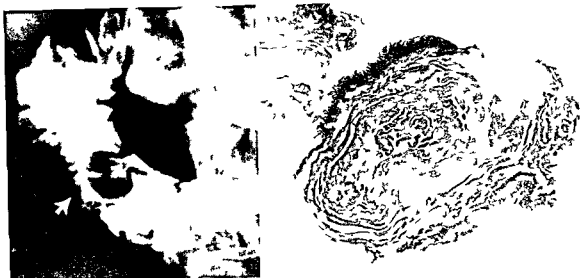


FIG 416 (A *Left*) Adenomatous polyp of the papilla of Vater. Note the sharply defined area of translucency produced by the tumor. (B, *Right*) Low power view of a cross section of the tumor.

of the three episodes of intestinal bleeding. Figure 415 C shows a cross section of the duodenal polyp.

BENIGN TUMORS OF THE PAPILLA OF VATER

Benign tumors of the papilla of Vater are very rare. Christopher⁴⁸ in 1953 reviewed 41 cases in the literature of benign tumors of the extrahepatic bile passages to which he added a case of his own of an adenoma of the ampulla of Vater. He stated that Bouisson in 1843 described a lipoma of the ampulla of Vater in a cadaver. The authenticity of some of the earlier reports was open to question. In only 6 cases was the ampulla of Vater definitely involved.

Baggenstoss⁴⁹ studied the duodenal papilla in 100 cases at postmortem. In 4 he found a polyp of the papilla. Calzavara⁵⁰ reported 2 cases of adenoma of the papilla of Vater. Baggenstoss found records of 25 polyps of the major papilla at the Mayo Clinic. They were incidental findings at autopsy. The polyps varied in size from 2 to 5 mm in diameter. They appeared as small fleshy, grayish or pink elevations that seemed to fill the orifice of the papilla. Two cases of benign papilloma of the

ampulla of Vater were described by Catell and Partek.⁵¹ They also included 4 cases of carcinoma of the papilla, apparently originating in a benign lesion of the ampulla.

A benign tumor of the papilla of Vater may be demonstrable in two ways. First, it may be recognized during the course of cholangiographic demonstration of the common duct, if the tumor intrudes within its lumen. This will be discussed and illustrated in the chapter dealing with cholangiography. Second, when the tumor of the papilla protrudes into the lumen of the duodenum, it may produce a translucent area within the confines of the barium filled segment. This is described in the following case.

The patient, A. D., male, aged 70, gave a 1 month history of progressive jaundice, abdominal pain, nausea, chills, fever, and a loss of 12 pounds during this period. The stools were clay colored. Physical examination revealed jaundice and an enlarged soft non-tender liver. The most significant findings were those revealed in the roentgenographic examination of the duodenum (Fig. 416 A). There was a sharply defined, rounded translucent area at about the anatomic position of the papilla. A diagnosis of a tumor of the papilla of Vater was made. A local resection of the tumor was performed.

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At operation, the duodenum was opened in the middle of the second portion, and a polyp, about 3½ inches long, was found originating at the junction of the first and the second portions but present within the second portion of the duodenum. A local removal of the polyp was performed. The base was ligated and sutured. The opening in the duodenum, which was longitudinal, was then sutured transversely.

Pathologic diagnosis. Adenomatous polyp of the duodenum.

Figures 415 B and C show the appearance of the polyp. In Figure 415 B, showing the external appearance of the polyp, one may note the ulcer which was the apparent cause



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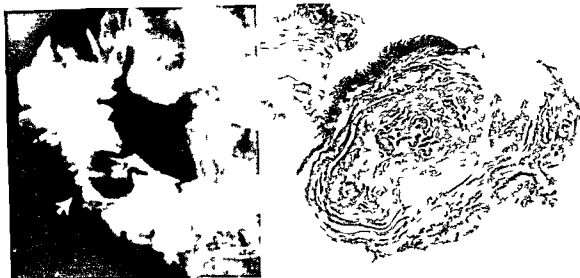


FIG 416 (A, *Left*) Adenomatous polyp of the papilla of Vater. Note the sharply defined area of translucency produced by the tumor (B, *Right*) Low power view of a cross section of the tumor

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The patient, A. D., male, aged 70, gave a 1 month history of progressive jaundice, abdominal pain, nausea, chills, fever, and a loss of 12 pounds during this period. The stools were clay colored. Physical examination revealed jaundice and an enlarged soft non-tender liver. The most significant findings were those revealed in the roentgenographic examination of the duodenum (Fig. 416 A). There was a sharply defined, rounded translucent area at about the anatomic position of the papilla. A diagnosis of a tumor of the papilla of Vater was made. A local resection of the tumor was performed.

Pathologic diagnosis Adenomatous polyp of the papilla of Vater (Figure 416 B shows a low-power cross section of the polyp)

A rare type of duodenal tumor which may be clinically significant and produce roentgenographic evidence of its presence is the enterogenous cyst. These cysts probably originate as fetal inclusions (Lewis and Thyng, *ibid*) and may be found anywhere in the intestinal canal. The cyst may be compared with a diverticulum of congenital origin which has been completely separated off from the lumen of the alimentary tract by obliteration of the area of communication. The cysts of the duodenum may lie directly beneath the mucosa or between the muscle layers of the intestinal wall or beneath the serosa. When present between the layers of the mesentery it may be a precursor of a mesenteric cyst. This phase of the subject will be described later in connection with a discussion of that particular subject.

The duodenal cyst has 5 main characteristics:

- 1 It is intimately adherent to some part of the duodenal wall

- 2 The wall of the cyst is composed of all the layers of the intestine. However, increased pressure within the cyst may produce some necrosis of the mucosa with partial denudation. The muscularis may be thin and attenuated.

- 3 The cyst contains a fluid that is usually clear and may be transparent like a hydrocele. It represents the secretion of its own epithelial lining. At times the fluid may be cloudy or dark due to some hemorrhage within it.

- 4 The cyst is completely separated from the lumen of the duodenum itself without any trace of its original communication.

- 5 It may compress and narrow the duodenal lumen, causing obstructive symptoms such as vomiting.

In the case reported by Waugh⁵² the enterogenous cystic mass was found in a 19-day-old female. It was the size of a tangerine and intimately connected with the

posterior wall of the duodenum. It had produced obstruction by compression of the first and the second portions of the duodenum. The enterogenous cyst of the duodenum reported by Gardner and Hart⁵³ was found in a 15-year-old girl who had complained of intermittent attacks of abdominal pain and vomiting. The cystic mass was within the wall of the duodenum. Aspiration yielded a thin clear slightly greenish fluid.

Orgias⁵⁴ reported an enterogenous cyst of the duodenum in an infant 7 weeks old. It was intimately attached to the pylorus and the first portion of the duodenum, which was partially obstructed. Booher and Pack,⁵⁵ in reviewing the literature up to the time of the publication of their paper in 1946, found records of 12 enterogenous cysts of the duodenum, to which they added a detailed description of two operatively confirmed cysts of their own. An additional case (the fourteenth in the world literature) was reported by Shallow, Wagner and Manges.⁵⁶ The cyst involved the second portion of the duodenum and was submucosal in location. It occurred in a 12-year-old male. Of the 14 cases, 10 were encountered between the time of birth and the age of 4 months. The oldest was the 15-year-old patient already referred to, which was described by Gardner and Hart.

The duodenal cyst described by Peple,⁵⁷ however, occurred in a female, aged 69. The cyst was within the anterior wall of the duodenum. It consisted of two thin-walled cysts shaped like a dumbbell and was removed by local enucleation. It had compressed the lumen of the first portion of the duodenum. The second portion of the duodenum was invaded by the enteric cyst described by Lorber and Machella.⁵⁸

The roentgen deformity produced by the cyst depends on two factors: (1) its location and (2) the degree of occlusion of the lumen of the duodenum. It may produce a rounded translucent area occupying most of the duodenal bulb (Peple).⁵⁷ The lumen of the duodenal bulb may be narrowed as a

result of a concave defect of the greater curvature border of the bulb because of the submucosal localization of the cyst in this region (Booher and Pack⁵) In the second portion of the duodenum, the cyst may produce a sharply defined rounded intraluminal area of translucency (Booher and Pack,⁵ Lorber and Machella⁶) However, the cyst may also produce an irregularly outlined ill-defined deformity of the medial border of the second portion of the duodenum (Shallow, Wagner and Manges⁶) There are no roentgen characteristics which would help to differentiate a cyst of the duodenum from other types of tumors It is possible that the diagnosis might be suspected if a deformity of the duodenum which might conceivably be produced by a cyst was found in early infancy or childhood

An interesting case of two lipomas of the second portion of the duodenum was reported by Fawcett, Bolton and Geever.⁷ An additional lipoma was found in the prepyloric portion of the stomach Preoperative roentgen examination revealed a translucent area in the pars pylorus and an independent translucency within the confines of the second portion of the duodenum The diagnosis of multiple lipomas was established on the basis of histologic study of the resected tumors

Benign tumors of the third portion of the duodenum are extremely rare Allison and Babcock⁶⁰ in the roentgenographic examination of a patient with a history of recurrent episodes of melena discovered a sharply defined rounded translucent area in the third portion of the duodenum Operation revealed a soft pedunculated lipoma as the cause of the roentgen deformity The authors found in the literature records of less than 20 lipomas of the duodenum most of them discovered incidentally at autopsy

Although benign tumors of the duodenum are rare enough a polyp of the third portion of the duodenum is most unusual The following is a completely documented case



FIG 417 Adenomatous polyp of the third portion of the duodenum (A Top) Note the intraluminal translucency produced by the polyp (B, Bottom) Appearance of the excised polyp Note the close correlation of the roentgenographic deformity and the polyp

E G male aged 63 The patient gave a 2 month history of anorexia epigastric pain and increasing weakness He also complained of regurgitation and pyrosis after meals He had lost an undeterminable amount of weight during the preceding few months Physical examination of the abdomen was essentially negative Roentgenographic examination (Fig 417 A) showed a fairly well circumscribed area of translucency in the third portion of the duodenum having the characteristics of a polyp This was confirmed at operation A duodenostomy was done and a pedunculated polyp 3 x 5 cm in size was excised

Pathologic diagnosis Adenomatous polyp of the duodenum Figure 417 B shows the appearance of the polyp Note how closely the translucent area in the roentgenogram corresponds to the anatomic appearance of the polyp



FIG. 418 (A, *Left*) Enormous dilatation of the entire duodenal curve resulting from the obstruction produced by a fibromyoma. (B, *Right*) Same patient. Appearance at 24 hours, showing the retention in the stomach and in the dilated duodenal curve.

An example of the extreme degree of obstruction of the duodenal curve which may result from the presence of a benign tumor is illustrated by the following case:

I. R., male, aged 47. This patient had been complaining for a period of 8 months of attacks of vomiting, abdominal distress and mild, dull, epigastric pain radiating slightly from right to left and occurring about once a week. The attacks were accompanied by the vomiting of a greenish-yellow fluid. There was no blood in the vomitus or in the stools. The patient was not jaundiced. He had lost 8 pounds in the preceding 8 months.

At operation, examination revealed telescoping and twisting of the small intestine into itself. The intestine was dilated enormously to about three times its normal size and was dark red, almost bluish in color. On reducing the intussusception it was made up entirely of jejunum. The furthest portion was revealed as a hard, nodular, grayish-white mass extending through the wall; it was about the size of a lemon and was apparently malignant. The intussusception was reduced with difficulty. Hot applications were applied to the intestine until the color approached normal. The mass was isolated and excised with cautery.

Gross description of the resected specimen was as follows: The specimen consisted of a portion of small intestine. Situated in its wall and projecting into its lumen was a globular tumor about 4 cm. in diameter. Microscopic examination showed the tumor to be situated in the muscular coat of the intestinal wall. The tumor was composed of smooth muscle fibers and fibrous tissue. The mucosa and submucosa showed no pathologic changes except some congestion. The diagnosis was fibromyoma of the small intestine.

Roentgen examination prior to operation (Fig. 418, A) showed the stomach and the duodenal bulb to be normal. The entire duodenal curve was enormously dilated, extending up to about the duodenojejunal junction. At 24 hours (Fig. 418, B) there was considerable retention of barium in the stomach and in the dilated transverse portion of the duodenum. The roentgen diagnosis was Obstruction of the transverse portion of the duodenum at about its junction with the jejunum, organic in origin and probably due to a tumor in this region. As noted above the tumor was a benign fibromyoma.

An unusual type of obstruction of the duodenum of extrinsic origin is that reported by Baird.¹¹ In his case obstruction of the third portion of the duodenum, with consid-

erable dilatation of the region proximal to it had resulted from the pressure of an aneurysm of the abdominal aorta. This was verified at operation.

Rarely, marked obstruction with dilatation of the duodenum may result from the pressure of an extraduodenal new growth. This is illustrated by the following case.

H. G., male, aged 33. After a long history of epigastric pain associated with nausea and vomiting, exploratory laparotomy was done for an abdominal mass. The report was as follows: There was a large mass filling the retroperitoneal space and the root of the mesentery, the greatest portion appearing to be in the region of the duodenojejunal junction filling the retroperitoneal space immediately below this region.

Microscopic diagnosis of a specimen removed at operation showed the tumor to be a metastatic carcinoma, probably seminoma.

Roentgen examination about 2 years later (Fig. 419), representing the appearance at 6 hours, revealed the stomach to be of considerably increased size. The duodenal bulb was enormously dilated. In addition, there was considerable dilatation of the proximal region of the second portion of the duodenum. The enormous size of the duodenum was well

seen in the 6 hour observation. There was a trace of barium in the stomach at 24 hours. The evidence indicated the presence of an obstructive lesion just beyond the duodenal bulb, which was responsible for the huge size of the duodenal bulb and the gastric retention at 24 hours. As noted at operation, this obstruction was produced by the pressure of a metastatic carcinoma, probably a seminoma.

DUODENAL TUBERCULOSIS

Matthews, Delaney and Dragstedt⁶⁷ found 105 cases of tuberculosis of the duodenum in a survey of the literature. These were part of a generalized intestinal tuberculosis. In addition they found reports of 18 cases of localized duodenal tuberculosis, in only 6 of which, however, was the diagnosis verified histologically. To these cases the authors added a personal case of hyperplastic tuberculosis of the duodenum involving the first portion. Histologic study showed a characteristic tubercle with giant cells, small round cells and fibroblasts.

Marked dilatation of the entire duodenum up to the duodenojejunal flexure as an



FIG. 419 Obstruction of the duodenum due to an extraduodenal tumor

indirect result of intestinal tuberculosis was reported by Hochstetter⁶³ At operation, an old tuberculous peritonitis was found About three fingers' breadth below the ligament of Treitz a severe stenosis of the intestine was found, due to a sharply constricting strand of connective tissue

The roentgenographic appearance of tuberculosis of the duodenum is shown in the following case

M D, female, aged 27 Two years prior to admission to the hospital the patient noted the gradual onset of right upper quadrant pain which eventually became so severe that an exploratory laparotomy was done at another hospital on the assumption that she had gall-bladder disease The surgeon reported that he found a severe peritonitis There was tuberculosis of the mesenteric nodes, as well as of the liver and the spleen Enlarged nodes were found on the posterior wall of the duodenum The diagnosis of tuberculosis was confirmed by microscopic examination Two months later she was referred to Bellevue Hospital with a diagnosis of tuberculous peritonitis

Physical examination revealed no recognizable pathology of the lungs The liver was palpable 10 cm below the right costal margin It was firm and slightly tender The spleen was palpable from 2 to 3 cm below the left costal margin There was a healed right upper rectus scar The temperature was 101.2° F While under observation the patient became increasingly lethargic and complained of intermittent frontal headache Clinical and laboratory investigation supported the diagnosis of tuberculous meningitis There was also rigidity and tenderness in the right upper quadrant The patient developed mild convulsions and became disoriented, and death supervened

Roentgenographic examination of the lungs revealed no evidence of tuberculosis Very significant findings were noted in the roentgenographic examination of the duodenum Figure 420 A shows the appearance of the duodenal curve promptly after the ingestion of the barium suspension Note the marked abnormality of the mucosal pattern of this segment with some vaguely outlined sinus tracts extending from the lateral border of the second portion of the duodenum These sinus tracts are more clearly noted in the examination 2 hours later (Fig 420 B) and also at 6 hours (Fig 420 C), at which time there is residual barium in the duodenum, although

none is present in the stomach From a survey of these three views it was quite obvious that there was a serious pathologic process involving the first and the second portions of the duodenum and that the lesion was associated with sinus tracts It was particularly because of the presence of these unusual sinus tracts that I ventured the opinion that the lesion was tuberculous The appearance, as far as the sinus tract formation is concerned, is in that respect at least similar to the roentgenographic appearance in the case of tuberculosis of the pylorus which was previously described (Fig 280).

Autopsy revealed generalized fibrocaceous and calcific tuberculosis of lymph nodes There was tuberculous meningitis, tuberculosis of the liver and the spleen and a calcified process in the right upper lobe of the lung

Particularly significant in this case were the findings in the examination of the digestive tract The esophagus and the stomach were normal The duodenum for a distance of about 7 cm from the pylorus was thickened and showed marked trabeculation of the mucosa The posterior wall of this portion of the duodenum was adherent to the pancreas A few lymph nodes throughout this area averaging about 3 to 7 mm in diameter showed caseation as well as calcification The remaining portion of the duodenum, as well as the jejunum and the ileum, were normal The colon was also normal except for a few diverticula of the cecum and the ascending portion

Microscopic examination of the involved duodenum showed marked fibrosis of all the layers as well as a severe chronic inflammatory cellular reaction Occasional sinus tracts were visualized, lined by granulations and caseous tissue infiltrated by lymphocytes

The gross appearance of the mucosa of the involved duodenum is shown in Figure 420 D Note the absence of normal mucosal folds, as well as the scarred and depressed areas Figure 420 E shows a sinus tract in the wall of the duodenum lined with tuberculous granulation tissue Figure 420 F shows infiltration with lymphocytes and some caseation necrosis and fibrosis in the submucosa of the duodenum

The roentgenographic findings in this case of abnormalities of the duodenum associated with sinus tracts upon which a diagnosis of probable tuberculosis was made were therefore fully confirmed by the pathologic studies

A tuberculous lesion may cause obstruction of the duodenum

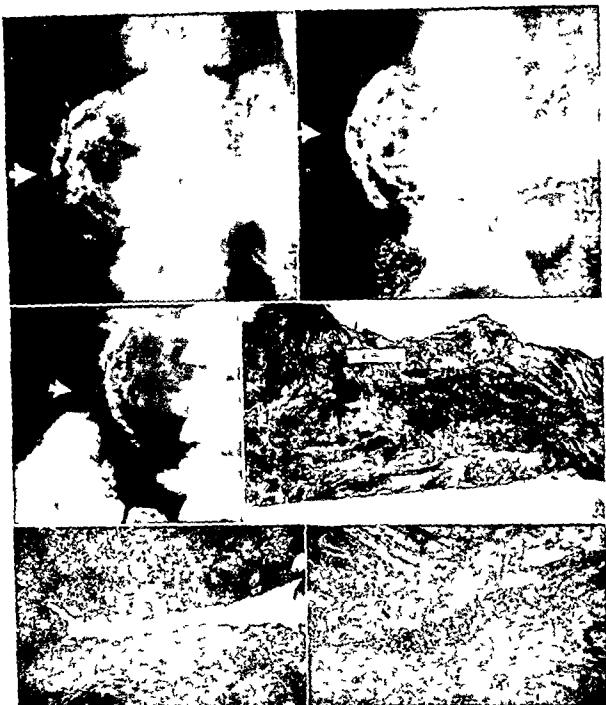


FIG. 420 Tuberculosis of the duodenum (A, *Top left*) Appearance of the duodenum promptly after the ingestion of barium. Note the abnormal mucosal pattern of this segment and a few faintly outlined sinus tracts which are seen much more clearly in B (*Top right*), taken 2 hours later and in C (*Center, left*) the appearance noted at 6 hours (D *Center right*) Note the absence of normal mucosal folds of the involved portion of the duodenum (E *Bottom left*) Photomicrograph showing sinus tract in the wall of the duodenum lined with tuberculous granulation tissue (F, *Bottom right*) Photomicrograph showing infiltration with lymphocytes and some caseation necrosis and fibrosis in the submucosa of the duodenum

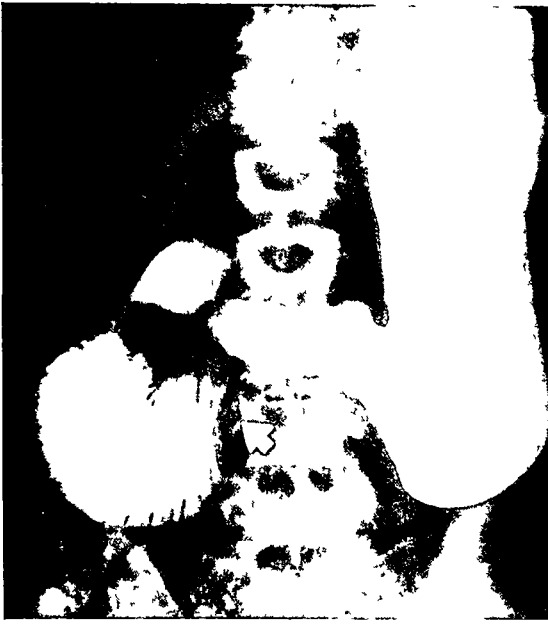


FIG 421 Dilatation of the second and the third portions of the duodenum secondary to constriction by a tuberculous band of tissue

C L, female, aged 34 The patient gave a history of the sudden onset of vomiting 3 weeks prior to admission to the hospital From then on the vomiting persisted, occurring from a few minutes to 1 hour after every meal, regardless of the type of food The vomitus was stained with bile but it did not

contain any blood There was no actual abdominal pain, but a slight soreness accompanied the vomiting She stated that the stools were black on a few occasions She lost 40 pounds in the preceding 3 weeks She had an episode of vomiting 4 years previously which lasted for 1 or 2 days

Physical examination revealed a malnourished white female who was markedly dehydrated The abdomen was soft and nontender There was a hard, ill-defined, slightly movable mass on the left side at the level of the umbilicus which transmitted pulsations of the aorta

Roentgenographic examination (Fig 421) revealed considerable dilatation of the second and the third portions of the duodenum There was sharp demarcation of the barium at the distal end of the visualized third portion of the duodenum, with practically none of the barium escaping beyond this zone There was also evidence of a pressure defect with displacement of the greater curvature border of the pyloric arm of the stomach The diagnosis was of an extrinsic mass, producing occlusion of the third portion of the duodenum

Operation revealed the following "The patient had numerous grayish-white rice-kernel-sized nodules over the peritoneum and the mesentery and the small bowel In two areas, one overlying the third portion of the duodenum and immediately anterior to the vertebral column, there was a dense yellowish-gray gristly band of tissue which encircled the third portion of the duodenum for approximately

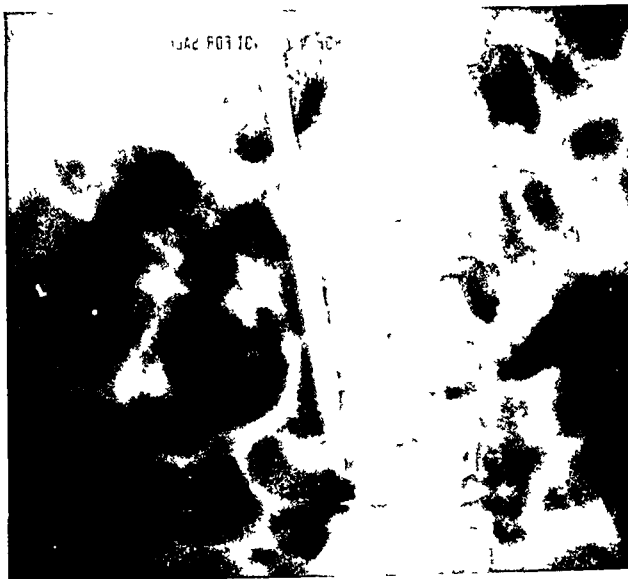


FIG 422 Thermometer in the duodenum

three quarters of its circumference. On section the interior of this band appeared to be caseous. There was a similar band just distal to the ligament of Treitz.

The band was dissected from the third portion of the duodenum and removed as a specimen. In addition, three other areas of tuberculous inflammatory reaction were removed for microscopic examination. Following complete mobilization of the third portion of the duodenum it was seen that this portion of the bowel could be straightened out, whereas formerly it had been linked over the band. However, when replacing the duodenum in the abdominal cavity it was seen that again the duodenum tended to fold on itself in this area. Accordingly, it was decided that a by-pass should be done and a duodenojejunostomy was performed using the first portion of the duodenum and a loop of jejunum approximately 10 to 12 cm below the ligament of Treitz.

The pathologic findings were reported as follows:

Macroscopic examination: Specimen received, fixed in Bouin's solution, consists of three firm, irregularly oval shaped coarsely granular surfaced pieces of tissue, the largest measuring 2 x 1 x 0.6 cm. On section the surface is homogeneous.

Microscopic examination: Three sections reveal nodules of granulation tissue. These have multiple large centers of caseous necrosis. The margin of these centers is composed of epithelioid cells and Langhans giant cells. Peripherally, there is a chronic inflammatory reaction to scar tissue.

In this case, therefore, there was a generalized tuberculous peritonitis with a dense band of tissue which partially encircled the transverse portion of the duodenum, causing the marked obstruction noted roentgenographically and corroborated surgically.

Rarely, a foreign body may be stuck in the duodenum and may require removal surgically. This is illustrated by the following case:

J. R., female, aged 13. While on the ward of the hospital being treated for a pulmonary and gynecologic condition, the child swallowed a thermometer.

At operation the thermometer was found in the duodenum. It was milked back into the stomach. An incision was then made through the pars media of the stomach and the thermometer removed.

The thermometer is shown in the roentgenogram (Fig. 422).

REFERENCES

1. Jefferson, G. Carcinoma of the suprapapillary duodenum causally associated with pre-existing simple ulcer: report of a case, and an appendix of 30 collected cases, *Brit J Surg* 4: 209, 1916.
2. Hoffman, W. J. and Pack, G. T. Cancer of duodenum: a clinical and roentgenographic study of eighteen cases, *Arch Surg* 35: 11, 1937.
3. Fenwick, W. S. Primary carcinoma of the duodenum, *Edinburgh M J* 10: 309, 1901.
4. Rolleston H. D. A clinical lecture on carcinomatous stricture of the duodenum, *Lancet* 1: 1121, 1901.
5. Meyer, J. and Rosenberg, D. H. Primary carcinoma of the duodenum: report of four cases with a review of the literature, *Arch Int Med* 47: 917, 1931.
6. Bookman, M. R. Carcinoma in the duodenum originating from aberrant pancreatic cells, *Ann Surg* 95: 464, 1932.
7. Stewart, H. L. and Lieber, M. M. Carcinoma of the suprapapillary portion of the duodenum, *Arch Surg* 35: 99, 1937.
8. Allen, C. I. Primary carcinoma of the duodenum with a report of eleven cases, *Am J Surg* 40: 89, 1938.
9. Fodden, J. H. Duodenal spread of pyloric carcinoma, *Brit J Cancer* 2: 239, 1948.
10. Zininger, M. M. and Collins, W. F. Extension of carcinoma of the stomach into the duodenum and esophagus, *Ann Surg* 130: 557, 1949.
11. Soper, H. W. The roentgen ray diagnosis of lesions of the small intestine, *Am J Roentgenol* 22: 107, 1929.
12. Mateer, John G. and Hartman, F. W. Primary carcinoma of the duodenum, *JAMA* 99: 1853, 1932.
13. Swenson, P. C. and Levin, A. G. Primary carcinoma of the duodenum: case report, *Am J Roentgenol* 31: 204, 1934.
14. Lachapele, A. and Dumon, G. Sur deux cas de stenose duodenale, *Bull et mem Soc radiol, med France* 25: 286, 1927.
15. Claiborn, I. N., and Dobbs, W. C.

- Carcinoma of the third segment of duodenum, report of a case, *Surgery* 4 97, 1938
- 16 Prey, D, Foster, J M, and Dennis, W Primary sarcoma of the duodenum, *Arch Surg* 30 675, 1935
- 17 von Salis, H W Über das Sarcom des Duodenums ins besondere des Myosarkoms, *Deutsche Zeitschr Chir* 160 180, 1920
- 18 Andersen, D H, and Doob, E F Leiomyosarcoma of the duodenum, *Arch Path* 16 795, 1933
- 19 Silverstone, M Sarcoma of the duodenum, report of a case, *Brit J Surg* 22 443, 1934
- 20 Seymour, W J, and Gould, S E Leiomyosarcoma of the duodenum, *Am J Cancer* 28 572, 1936
- 21 Foshee, J C, and McBride, W P L Leiomyosarcoma of the duodenum, *J A M A* 12 2497, 1939
- 22 Mayo, C W Malignancy of the small intestine, *West J Surg* 48 403, 1940
- 23 Henning, B H, and Garland, L H Leiomyosarcoma of the duodenum, *Radiology* 37 353, 1941
- 24 McCullough, Kendrick Leiomyosarcoma of the duodenum, *New York State J Med* 44 1248, 1944
- 25 Shackelford, R T, Fisher, A M, and Firor, W B Duodenal tumor of unusual character, *Ann Surg* 116 864, 1942
- 26 Pohlandt, K Die rontgenologische Diagnose und Differentialdiagnose der Tumoren des Bulbus duodeni, *Fortschr Geb Rontgenstrahlen* 43 337, 1931
- 27 Sielaff, Hans-Jürgen Der Rontgenbefund von multiplen Melanometastasen in Duodenum und Dunndarm, *Fortschr Geb Rontgenstrahlen* 71 592, 1949
- 28 Carman, R D Hemangioma of the duodenum, *Am J Roentgenol* 8 481, 1921.
- 29 Golden, R Nonmalignant tumors of the duodenum report of two cases, *Am J. Roentgenol* 20 405, 1928
- 30 Waters, C A The roentgenologic diagnosis of papilloma of the duodenum, *Am J Roentgenol.* 24 554, 1930
- 31 Brdiczka, I G Die Tumoren des Bulbus Duodeni und ihre rontgenologische Diagnose, *Rontgenpraxis* 3 625, 1931
- 32 Raiford, T S Tumors of the small intestine their diagnosis, with special reference to the x-ray appearance, *Radiology* 16 253, 1931
- 33 Bonomini, B Ascaridi nel bulbo duodenale, *Riv radiol e fis med* 6 270, 1931
- 34 Venables, J F A case of small intestine obstruction due to Hodgkin's disease, *Guy's Hosp Rep* 78 377, 1928
- 35 Lubarsch, O Ueber den primären Krebs des Ileum nebst Bemerkungen über das Gleichzeitige Vorkommen von Krebs und Tuberkulose, *Virchows Arch path Anat* 111 280, 1888
- 36 Oberndorfer, S Karzinoide Tumoren des Dunndarms, *Frankfurt Ztschr Path* 1 426, 1907
- 37 Masson, P La glande endocrine de l'intestin chez l'homme, *Compt rend Acad Sc* 158 59, 1914
38. Ariel, I M Argentaffin (carcinoid) tumors of small intestine, *Arch Path* 27 25, 1939
- 39 Porter, J E, and Whelan, C S Argentaffine tumors, *Am J Cancer* 36 343, 1939
- 40 Ritchie, G, and Stafford, W. T Argentaffin tumors of gastrointestinal tract, *Arch Path* 38 123, 1944
- 41 D'Ingianni, V Carcinoid of the appendix with metastasis, *New Orleans M & S J* 99 158, 1946
- 42 Cruickshank, B, and Cunningham, A W B The carcinoid tumor, a review of 17 cases, *Edinburgh M J* 56 196, 1949
- 43 D'Albora, J B, and Ingegno, A P Carcinoid tumors of the small intestine, *Gastroenterology* 10 310, 1948
- 44 Reid, D R K Argentaffinoma of the gastrointestinal tract, *Brit J Surg* 36 130, 1948
- 45 Brown, C H, Bissonnette, R P, and Steele, H H Argentaffine tumors of the gastrointestinal tract, *Gastroenterology* 12 225, 1949
- 46 Grimes, O F, and Bell, H G Carcinoid tumors of the intestine, *Surg, Gynec & Obst* 88 317, 1949
- 47 Pearson, C M, and Fitzgerald, P J Carcinoid tumors a re-emphasis of their malignant nature, *Cancer* 2 1005, 1949
- 48 Christopher, F Adenoma of the ampulla of Vater, *Surg, Gynec & Obst* 56 202, 1933
- 49 Baggenstoss, A H Major duodenal papilla, *Arch Path* 26 853, 1938
- 50 Calzavara, C Virchow's *Arch Path Anat* 141 221, 1895 (Quoted by Baggenstoss)
- 51 Cattell, R B, and Pyrttek, L J Premalignant lesions of the ampulla of Vater, *Surg, Gynec & Obst* 90.21, 1950

- 52 Waugh, O Congenital cyst of the duodenum, Surg, Gynec & Obst 37 785, 1923
- 53 Gardner, C E, Jr, and Hart D Enterogenous cysts of the duodenum Report of a case and review of the literature, J A M A 104 1809, 1935
- 54 Orgias, R Enterogenous cyst of the duodenum, Brit J Surg 31 90 1943
- 55 Booher, R J, and Pack, G T Cysts of the duodenum, Arch Surg 53 588 1946
- 56 Shallow, T A Wagner, F B and Manges W B Enterogenous cysts of the duodenum Surgery 21 532, 1947
- 57 Peple, W L Enterogenous cyst of duodenum report of case, Ann Surg 127 912, 1948
- 58 Lorber, S H and Machella T E Enteric cyst of the duodenum, Gastroenterology 10 892, 1948
- 59 Fawcett N W, Bolton, V L, and Geever, E F Multiple lipomas of the stomach and duodenum Ann Surg 4 524, 1949
- 60 Allison, R D, and Babcock, J H Lipomas of duodenum causing melena, Ann Surg 127 754, 1948
- 61 Baird, L W Obstruction of the third portion of the duodenum of unusual etiology report of a case, Radiology 27 235, 1935
- 62 Matthews, W B Delaney, P A, and Dragstedt, L R Duodenal tuberculosis a review of the literature and a report of a case of hyperplastic tuberculosis of the duodenum, Arch Surg 25 1055, 1932
- 63 Hochstetter F Duodenalstenose infolge alter Peritonitis tuberculosa, Fortschr Geb Rontgenstrahlen 29 176, 1922

